ARCHIVES OF OTOLARYNGOLOGY

VOLUME 43 MARCH 1946 NUMBER 3

COPYRIGHT, 1946, BY THE AMERICAN MEDICAL ASSOCIATION

TYMPANOSYMPATHECTOMY

A Surgical Technic for the Relief of Tinnitus Aurium

JULIUS LEMPERT, M.D.
NEW YORK

ETIOLOGICALLY, tinnitus aurium has remained unexplained. Though a number of theories have been advanced in the attempt to identify and explain the factors responsible for this distressing symptom complex, none of the theories has ever been proved either histologically or therapeutically.

During the last twelve years I have been conducting an extensive research study of the symptom complex known as tinnitus aurium in its relation to disease of the middle and the inner ear.

In lesions of the middle and the inner ear of nonsuppurative origin the following observations were made:

1. Some patients suffering from pure middle ear deafness as a result of stapedial ankylosis due to otosclerosis complain of tinnitus; in other patients of this type tinnitus is conspicuously absent.

2. Patients suffering from pure middle ear deafness due to otosclerosis who complain of tinnitus do so, as a rule, before their hearing for air-conducted sound drops to the 50 decibel level in the conversational frequencies. As the hearing loss progresses below this level, the intensity of the tinnitus gradually decreases, and soon the tinnitus completely disappears.

3. Some patients suffering from combined middle and inner ear deafness as a result of otosclerosis complain of tinnitus, while many others never do.

4. While many patients suffering from pure inner ear deafness of toxic origin complain of tinnitus, just as many are completely free from tinnitus.

5. Some patients suffering from inner ear deafness due to acoustic trauma complain of tinnitus, while others do not.

6. Patients suffering from congenital inner ear deafness do not complain of tinnitus.

From the Lempert Institute of Otology.

7. Tinnitus aurium when accompanying progressive degeneration of the cochlear nerve does not always disappear when clinical evidence of total loss of function in this nerve ensues; often it assumes even a much more aggravated and intensified form.

8. As confusing as the presence or the absence of tinnitus in deafness of nonsuppurative origin is to one in search of an explanation of this symptom complex, still more confusing is the fact that tinnitus aurium is often complained of by people with apparently normal middle and inner ears who are not deafened and never become deafened.

In lesions of the middle and the inner ear of suppurative origin the following observations were made:

1. Patients suffering from acute suppurative otitis media usually complain of tinnitus. Such tinnitus disappears, as a rule, when the acute infection of the middle ear resolves.

2. Occasionally the tinnitus does not subside following the resolution of the acute otitis media and persists indefinitely thereafter.

3. Patients left with chronic adhesive processes in a dry middle ear after the healing of subacute suppuration of the middle ear often complain of tinnitus which persists indefinitely.

4. Patients suffering from a chronic suppurative lesion with cholesteatoma limited to the epitympanum, without involving the tympanum proper to any great extent, and draining through a perforation in Shrapnell's membrane, sometimes complain of tinnitus. In such cases mastoidoatticotomy never has relieved the tinnitus.

5. Patients with either polypi or cholesteatoma filling the tympanum proper and with chronic suppurative otitis media of long standing draining through a perforated membrana tensa almost never complain of tinnitus.

6. Patients presenting clinical evidence of total loss of cochlear nerve function as a result of suppurative labyrinthitis never complain of tinnitus.

Of all the observations made, the most striking, and to me the most significant, was the extreme rarity of tinnitus in patients suffering from long-standing suppurative lesions with polypi and cholesteatoma involving the tympanum proper. This fact when coupled and contrasted with the observation that tinnitus aurium almost always accompanies acute otitis media suggested the following line of reasoning:

1. Tinnitus aurium may be the result of inflammation of some anatomic structure which is normally present within the tympanic cavity.

2. In acute otitis media, this anatomic structure becomes acutely inflamed, and tinnitus results.

3. If the inflammation within this anatomic structure subsides when the acute otitis media resolves, the tinnitus disappears.

4. When this structure is destroyed as a result of chronic suppuration of the middle ear, tinnitus is no longer possible.

In an attempt to prove or disprove the aforementioned suspicions I decided to inspect and investigate grossly with Zeiss magnifying binoculars all the anatomic structures normally known to be present and discernible within the tympanic cavity in two separate series of patients.

One series was limited to 100 patients complaining of tinnitus while suffering from pure middle ear deafness suspected to be due to otosclerosis. In the course of performing the fenestration operation, I carefully searched the tympanic cavity with magnifying glasses for grossly discernible pathologic changes in the anatomic structures known to be existing normally within it. Outside of fixation and immobility of the stapedial footplate, which were almost always encountered, and the frequently observed bony excresence in the niche of the oval window, the only other pathologic change frequently observed was abnormal congestion of the mucoperiosteum covering the cochlear promontory.

The other series, available for such investigation during a period of about six years, consisted of 60 consecutively encountered patients who were suffering from long-standing, chronic suppurative otitis media with cholesteatoma within the tympanum but who were not complaining of tinnitus aurium. During the performance of mastoidotympanectomy for the cure of the otitis media purulenta chronica the anatomy of the middle ear of every one of the 60 patients was carefully investigated for any unusual pathologic changes which could serve as a clue to the explanation of the consistent absence of tinnitus in all these patients.

With the aid of Zeiss magnifying glasses, the one constant change I was for the first time able to observe, in contrast to the usual varied and never constant pathologic conditions previously observed in this type of chronic infection of the middle ear when the patients were subjected to operation, was that the normally present branches of the tympanic plexus, which were readily observable within the mucoperiosteum covering the cochlear promontory in every one of the 100 patients who had clinical otosclerosis with tinnitus, were missing and not to be found in the mucoperiosteum of the promontory in any one of these 60 patients at the time of operation. Often not even a trace

of the bony grooves wherein the main branches of this plexus were lodged could be found in the cochlear promontory.

These opposite findings regarding the tympanic plexus in the two series of patients when correlated with the presence and the absence of tinnitus suggested the possibility of a relation between the histopathologic state of the tympanic plexus and the symptom complex of tinnitus.

I reasoned that perhaps an acutely or chronically irritated or inflamed tympanic plexus gives rise to tinnitus aurium, whereas death or absence of this plexus makes tinnitus aurium impossible. If this was

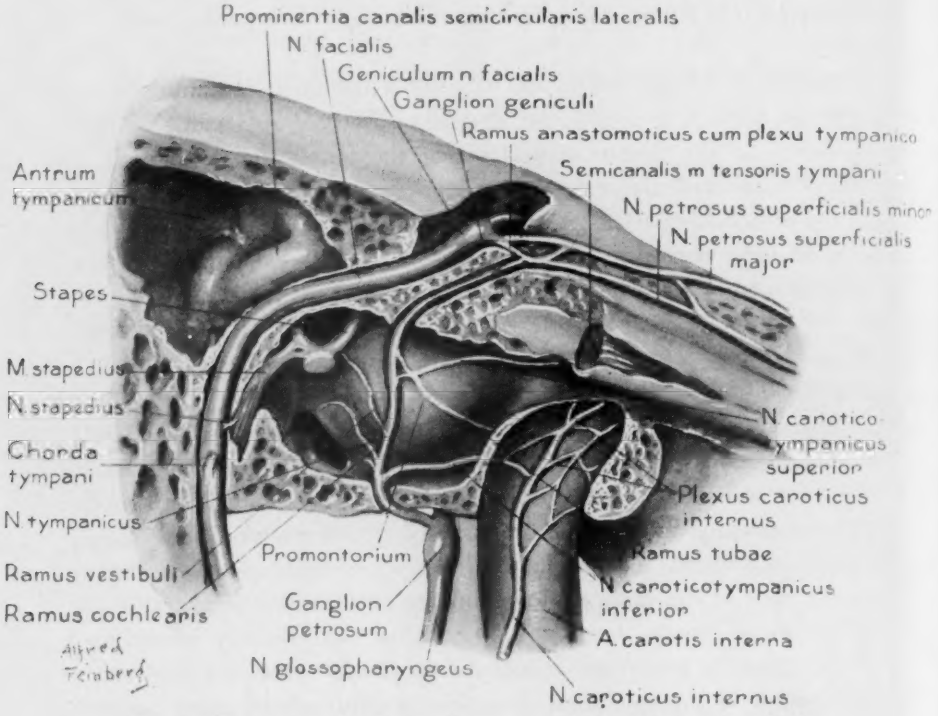

Fig. 1.—The tympanic plexus of nerves: origin, course and relations.

true, then the excision of the diseased tympanic plexus of a patient suffering from chronically persistent tinnitus aurium should result in cure of this symptom complex.

To prove the existence of a relation between tinnitus aurium and the tympanic plexus, it was obvious, the evidence would have to be both therapeutic and histologic.

In order to prove therapeutically the existence of such a relationship, I performed, in 1942, mastoidotympanectomy for chronic suppuration of the middle ear in a woman who was one of those extremely

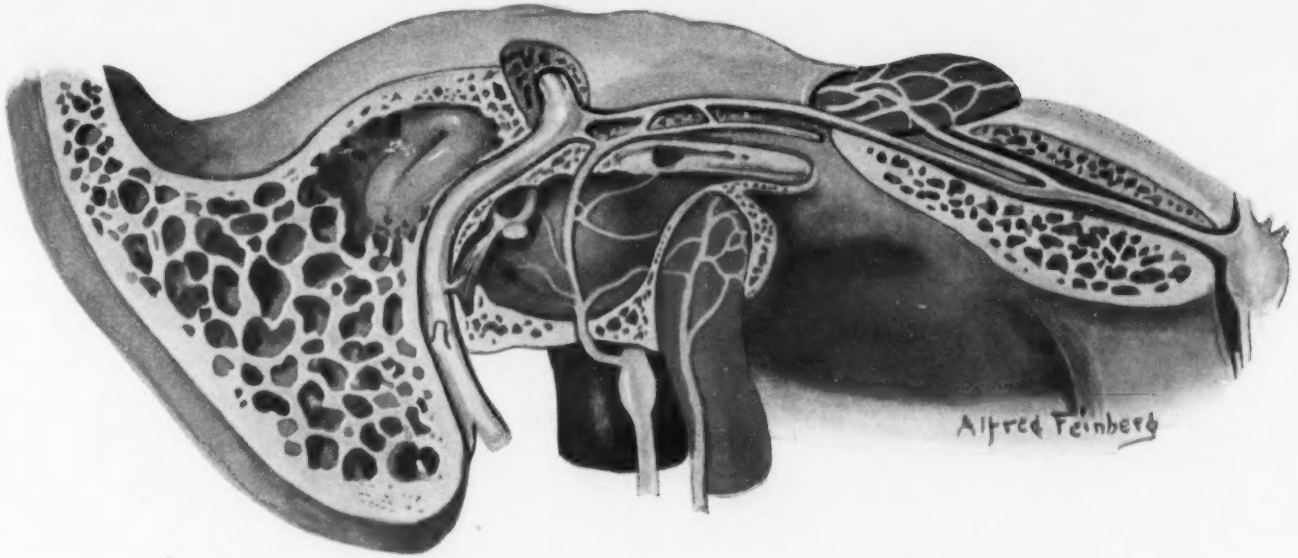

Fig. 2.—The tympanic plexus of nerves: origin, course and relations.

MCGM UNIV

rare patients in whom the suppuration of the middle ear, though chronic, was nevertheless accompanied by tinnitus aurium.

Though the otitis media purulenta chronica had been present for five years, the tympanic plexus was readily discernible with the Zeiss magnifying loop and apparently completely intact. In following my endaural technic of performing mastoidotympanectomy, after eradicating all the disease encountered, I carefully removed the entire mucous membrane lining the cochlear promontory and the rest of the tympanic cavity. Immediately following this operation, the tinnitus completely disappeared.

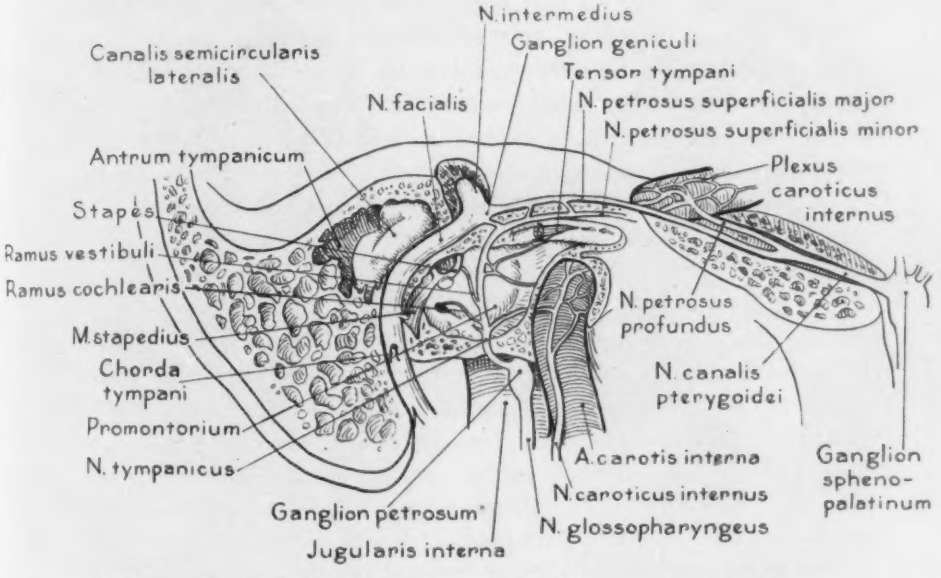

Fig. 3.—The tympanic plexus of nerves: origin, course and relations.

As encouraging as the findings, the therapy and the result were in this case, there was no way of being certain whether the mere removal of the pathologic processes occurring within the antrum, the aditus and the tympanic cavity would not have given the same result even if the mucoperiosteum with the tympanic plexus had been left intact.

In 1942 I performed mastoidoatticotomy on a woman who was suffering from chronic epitympanic suppuration of the right ear and complaining of severe tinnitus of seven years' duration in the same ear. Though I succeeded in giving the patient a dry ear, the tinnitus was not relieved. One year later, in 1943, I performed mastoidotympanectomy on the same ear, which was then perfectly dry, in order to inspect the

mucoperiosteum lining the middle ear cavity and observe whether the tympanic plexus was still present and discernible.

Inspection of the tympanum with Zeiss magnifying glasses revealed pale and thickened mucoperiosteum covering the cochlear promontory, with the tympanic plexus clearly discernible and visible. Though no pathologic processes requiring exenteration were encountered in the tympanic cavity, I carefully removed the entire mucoperiosteum covering the promontory, including the tympanic plexus from its grooves in the promontory. Immediately after this operation the tinnitus completely disappeared.

Since in this case no pathologic processes requiring exenteration within the tympanic cavity were encountered and since the only anatomic structures surgically removed were the somewhat thickened mucoperiosteum with the tympanic plexus, it was reasonable to assume that perhaps in this case there existed a definite relation between the tympanic plexus and the symptom complex of tinnitus aurium and that the disappearance of the tinnitus may have been the direct result of the excision of a diseased plexus.

OTONEUROLOGY

The plexus of nerves which can be observed in the mucoperiosteum lining the anterior portion of the tympanic cavity arises from the sensory fibers of the trigeminus, sympathetic and glossopharyngeal nerves. Jacobson's nerve, which arises from the glossopharyngeal nerve, is the most highly developed nerve in the tympanic plexus. This nerve contains isolated ganglion cells. It passes from the fossa jugularis through an opening in the inferior tympanic wall and, extending upward in a groove in the promontory, anastomoses with the small superficial petrosal nerve which arises from the trigeminus.

The sympathetic nerves of the lining membrane of the middle ear arise from the sympathetic plexus accompanying the carotid artery. Several small branches of this plexus enter the tympanic cavity through orifices in the carotid canal as the superior and inferior caroticotympanic nerves. These in conjunction with the small branches of Jacobson's nerve and the small superficial petrosal nerve arising from the trigeminus form the tympanic plexus in the anterior portion of the tympanic cavity. Arising from this plexus are smaller nerve branches which supply the entire lining membrane of the middle ear and in which one finds, in the regions of the fenestra vestibuli and the fenestra cochlearis, clusters of ganglion cells. Besides the latter, a delicate

ramifying network of nerve fibers is found which spreads partly above and partly below the vessels and forms ganglionic swellings where several fibers meet. A large branch arising from the tympanic plexus runs also to the cartilaginous eustachian tube.[1]

HOW CAN THE TYMPANIC PLEXUS BECOME RESPONSIBLE FOR TINNITUS AURIUM?

Theory: The anatomic and histologic structure of the tympanic plexus with the distribution of its branches, filaments and ganglionic

Figure 4

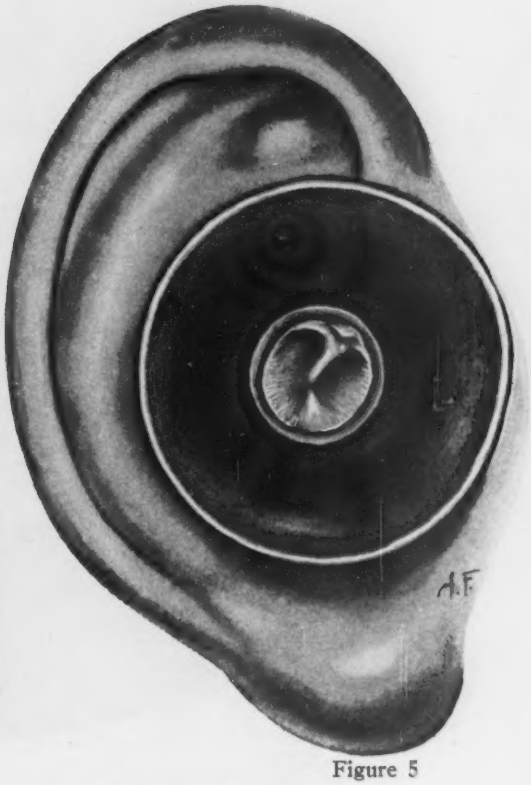

Figure 5

Fig. 4.—An incision is made in the membranous outer third of the superoposterior wall of the external auditory canal to permit a speculum with the widest fitting lumen to be introduced into the external auditory canal as near to the drum membrane as is possible under the circumstances encountered.

Fig. 5.—The speculum is shown in situ. Exposed to view and direct surgical approach is the tympanic membrane.

swellings is suggestive of a new plausible explanation of the symptom complex known as tinnitus aurium.

1. Politzer, A.: A Textbook of Diseases of the Ear for Students and Practitioners, ed. 5, London, Ballière, Tindall & Co., 1909, p. 36.

On the basis of the observations made in my research of this problem, I believe that in many cases tinnitus aurium may be a tonus impulse transmitted from the middle ear by the tympanic plexus to the inner ear. Acute, subacute or chronic ganglionitis involving some or all of the ganglionic swellings in the tympanic plexus may be

Fig. 6.—A semicircular incision is made in the cutaneous lining of the external auditory canal approximately 2 mm. external to the margin of the lower half of the tympanic membrane extending from about 3 to 9 o'clock.

responsible for tinnitus aurium. Such ganglionic disease may result from infection of the middle ear, venostasis in otosclerosis, trauma and toxic conditions of systemic origin. When ganglionitis resolves following the resolution of acute otitis media, the tinnitus subsides. If the

ganglionitis does not resolve and the ganglion cells undergo chronic inflammatory changes in subacute infections of the middle ear or in dry chronic adhesive lesions of the middle ear, the tinnitus persists. By the same token, if the inflammation of the ganglion cells results in suppuration and ultimate degeneration of the tympanic plexus follow-

Fig. 7.—With the aid of a periosteal elevator the cutaneous lining of the external auditory canal is subperiosteally elevated and the lower half of the annulus fibrosus of the tympanic membrane is disengaged from the sulcus tympanicus.

ing prolonged chronic suppuration of the middle ear with cholesteatoma, tinnitus aurium of ganglionic origin is no longer possible.

Whether the tonus impulse originates in the diseased ganglion cells of the tympanic plexus or whether the diseased ganglion cells increase

the intensity of, and act as the terminal source for, the transmission of otherwise normally not heard but normally present tonus impulses originating in the sensory fibers of the trigeminus, the sympathetic or the glossopharyngeal nerve before these enter the tympanic cavity to form the tympanic plexus is a subject for further investigation.

On the basis of such a theory one could perhaps attempt to explain the various types of tinnitus heard by these patients. One could reason

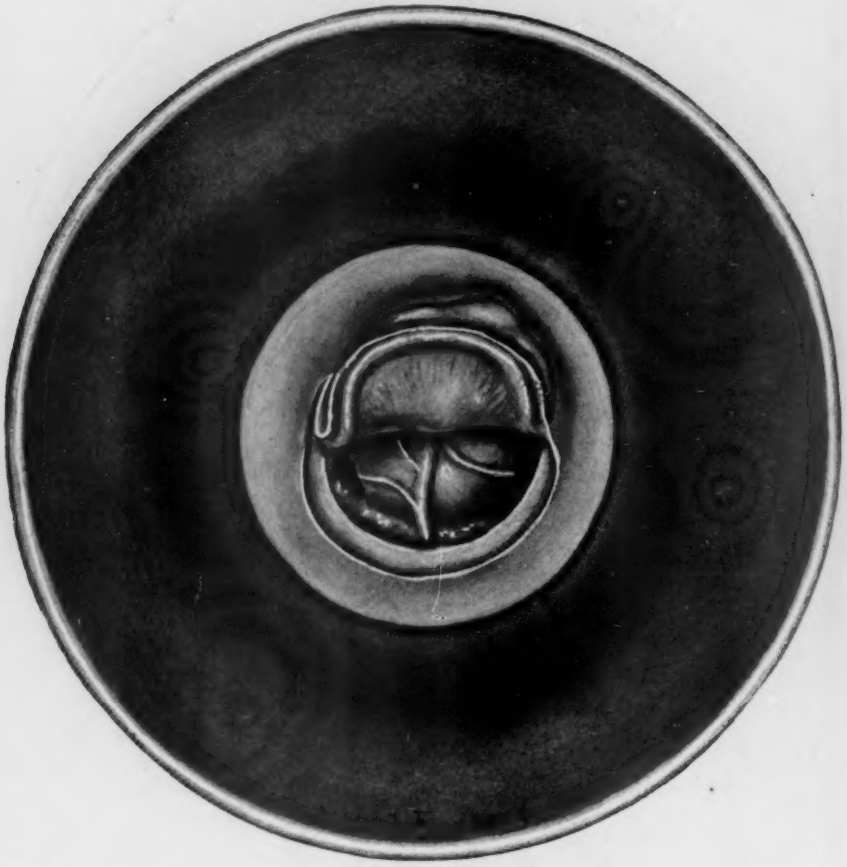

Fig. 8.—The lower half of the tympanic membrane with its annulus fibrosus is shown everted. Exposed to view is the mucoperiosteum covering the cochlear promontory with the tympanic nerve plexus clearly discernible with the aid of Zeiss magnifying glasses.

that when the ganglion cells in the region of the round window undergo some inflammatory or chemical or electric potential change, a tonus impulse is transmitted to the inner ear which is interpreted as high-pitched tinnitus by the organ of Corti. When ganglionitis occurs in

the region of the oval window, a tonus impulse is transmitted to the inner ear which is interpreted as low-pitched tinnitus. When ganglionitis involves the sympathetic caroticotympanic filaments coming from the carotid plexus and a tonus impulse is transmitted to the inner ear, it is interpreted as pulsating tinnitus by the organ of Corti.

When all the ganglion cells of the tympanic plexus are either acutely or chronically inflamed and more than one tonus impulse is

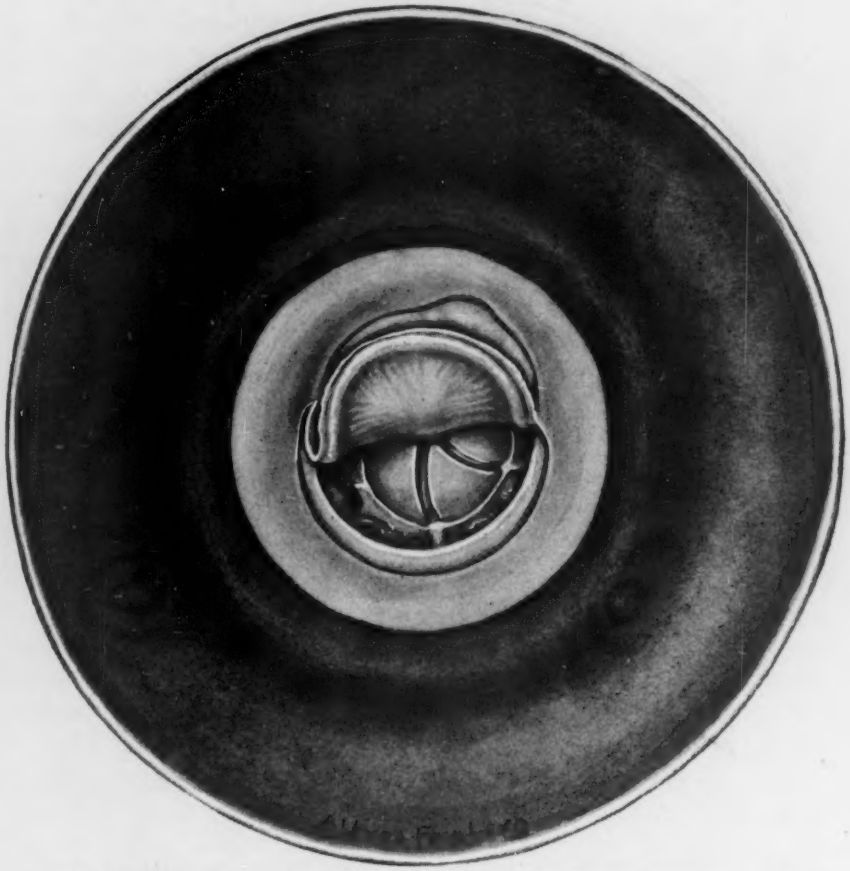

Fig. 9.—Exposed to view is the bony wall of the cochlear promontory, stripped of its mucoperiosteum, showing the bony grooves from which the tympanic plexus has been removed.

transmitted to the inner ear, a mixed type of tinnitus is heard. If this theory is correct, removal of the tympanic plexus should result in cure of tinnitus in those cases in which ganglionitis is the causative factor.

Armed with this theory, which was evolved as a result of observing gross pathologic changes at the operating table, I eventually decided

to attempt to relieve tinnitus aurium by excising the tympanic plexus. I have devised a technic for this purpose which can be performed without damaging the hearing acuity of such patients.

TECHNIC OF TYMPANOSYMPATHECTOMY

Anesthesia is obtained with the nitrous oxide–oxygen inhalation method. The operation consists of five procedures:

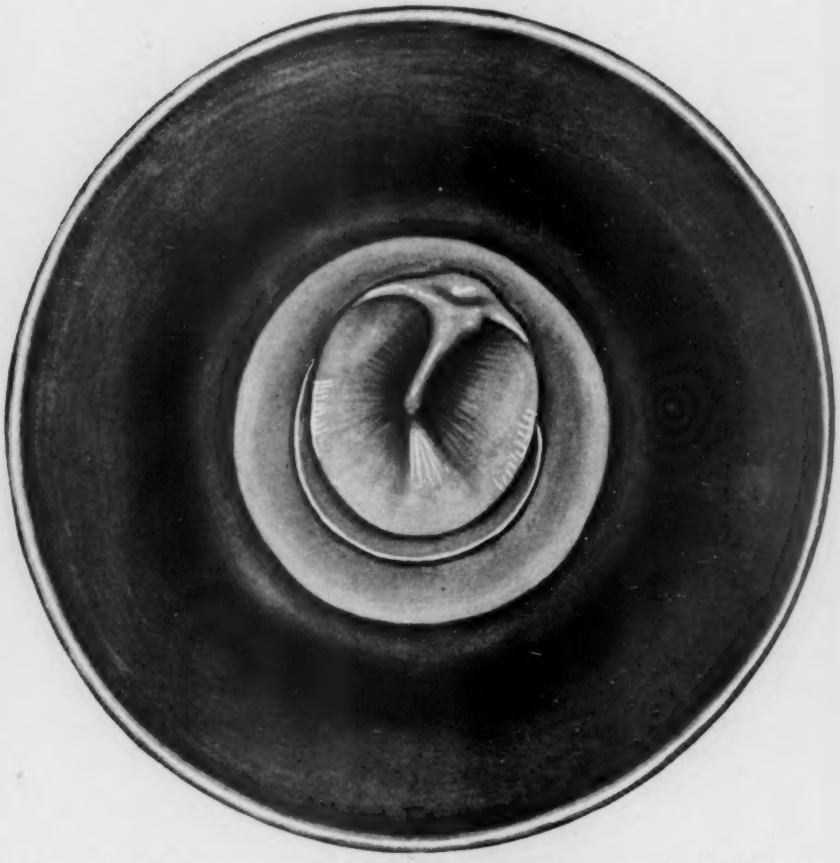

Fig. 10.—The tympanic membrane is now shown replaced to its normal position, with the annulus fibrosus framed once again in the sulcus tympanicus.

1. The outer membranous third of the external auditory canal is widened to permit the introduction of the widest possible ear speculum, through which the tympanic cavity will be made readily accessible to surgical intervention.
2. The lower one half of the annulus fibrosus of the tympanic membrane is disengaged from the corresponding portion of the sulcus tympanicus.

3. The disengaged lower half of the tympanic membrane is everted in order to expose to view the tympanic plexus.
4. The tympanic plexus is excised.
5. The disengaged portion of the annulus fibrosus of the tympanic membrane is reinserted into its normal position within the sulcus tympanicus.

OPERATIVE RESULTS

Tympanosympathectomy has been performed in 15 patients suffering from chronically persistent tinnitus aurium.

1. In 3 patients the tinnitus accompanied chronic adhesive middle ear deafness; the involved middle ears were dry and the drum membranes intact, with healed perforations: In all 3 patients the tinnitus disappeared.

2. In 5 patients tinnitus occurred in the presence of almost normal hearing: The tinnitus disappeared in 3 of them. In 2 it persists.

3. In 4 patients severe tinnitus occurred in the presence of total deafness due to complete degeneration of the cochlear nerve. In 3 of these the tinnitus vanished, and in 1 it persists.

4. Three patients had tinnitus accompanying a mixed form of middle and inner ear deafness: In 1 patient the tinnitus disappeared. In 2 it remained.

COMMENT

Since the removal of the tympanic plexus cannot be accomplished without the removal of the mucoperiosteum lining the cochlear promontory, the unknown factor still remaining to be determined is the part, if any, played by the removal of the mucoperiosteum proper in the 10 cases in which cure apparently was obtained by excision of this plexus.

The arterial blood supply of the mucous membrane lining the tympanic cavity is derived from (1) the tympanic branch of the internal maxillary artery, (2) the ascending pharyngeal branch of the external carotid artery, which runs over the promontory to the tegmen and anastomoses with the middle meningeal artery, (3) branches of the middle meningeal artery and (4) caroticotympanic branches of the internal carotid artery.

It has been demonstrated histologically, first by Politzer [2] and since by many other observers, that the blood vessels of the mucoperiosteum lining the cochlear promontory are accompanied by large connective

2. Politzer, A.: Ueber Anastomosen zwischen den Gefässbezirken des Mittelohres und des Labyrinths, Arch. f. Ohrenh. **11**:237-243, 1876.

tissue prolongations which penetrate funnel-shaped canals within the bony capsule of the cochlear promontory and communicate with the blood vessels of the bony capsule, through which they in turn communicate with the blood vessels of the endosteal membrane lining the perilymphatic labyrinth. On the basis of such histologic observations it is conceivable that vascular changes within the blood vessels of the tympanic mucoperiosteum lining the cochlear promontory may become an etiologic factor in the appearance of the symptom complex known as tinnitus aurium and that by the same token the severing of such a diseased arterial system which is accomplished by the removal of this mucoperiosteum could result in the abatement of tinnitus aurium. This phase of the problem is being further investigated.

SUMMARY

As a result of extensive research, a new theory is offered, suggesting that tinnitus aurium in many cases and under certain conditions may be due to a tonus impulse transmitted to the inner ear by diseased sympathetic ganglion cells of the tympanic plexus. Based on this new theory, tympanosympathectomy is recommended for the relief of tinnitus aurium.

The surgical technic by which tympanosympathectomy may be performed without disturbing the hearing acuity has been successfully employed.

Cognizance is taken of the fact that not all cases of tinnitus may be embraced by this new theory and of the fact that there is not yet any definite method of choosing the cases to which this theory definitely applies; it is, nevertheless, interesting that, of 15 patients on whom tympanosympathectomy was performed, 10 are now completely free from tinnitus. Of course, patients submitted to this surgical treatment should be those who complain of severe, continuous and persistent tinnitus of at least one year's duration. Patients who have to concentrate to hear their tinnitus were not subjected to this surgical treatment; rather those patients who complained that they could not concentrate on anything else because of their tinnitus were chosen.

To supplement the therapeutic proof of this theory, the tympanic plexus is now being histologically investigated. If ganglionitis can be repeatedly demonstrated histologically within the tympanic plexus in cases of tinnitus, conclusive substantiation of the new theory will be at hand.

OTITIS EXTERNA

Clinical Observations and Microbiologic Flora

LIEUTENANT JEROME T. SYVERTON (MC)*

CHIEF PHARMACIST'S MATE WILLIAM R. HESS
AND
LIEUTENANT (jg) JOHN KRAFCHUK (MC)
UNITED STATES NAVAL RESERVE

With the Assistance of

Pharmacist's Mate Second Class E. H. Josselyn
AND
Pharmacist's Mate First Class G. H. Freyvogel Jr.

THE high incidence and the widespread distribution of otitis externa (otomycosis, myringomycosis, fungous infection of the ear) among military personnel in tropical or subtropical climes, and on their return to the United States, make this clinical entity a medical problem of importance. More commonly considered to result from fungous infection, otitis externa is regarded by some authors and practitioners to have bacteria as primary or secondary causative agents. Others ascribe the pathologic changes to allergy or to seborrheic dermatitis or eczema.

We became interested in the etiologic problem when it was found that a diagnosis of otitis externa had been made for 17 (8 per cent) of 241 men within eight months after their arrival on Guam, in the Marianas. These men, free from "ear trouble" on departure from the United States, lived and worked under excellent hygienic conditions. The pathologic changes seen on otoscopic examination suggested bacterial infection. When preliminary bacteriologic studies [1] supported this impression, an investigation was planned to determine whether the microbiologic flora in exudates from the ears of patients with otitis externa included fungi and to learn what bacteria were present. Accordingly, 50 men presenting 72 ears diagnosed clinically as having the disease were studied.

From United States Naval Medical Research Unit no. 2.

*Now Associate Professor of Bacteriology, University of Rochester School of Medicine and Dentistry, Rochester, N. Y.

This article has been released for publication by the Division of Publications of the Bureau of Medicine and Surgery of the United States Navy. The opinions and views set forth are those of the writers and are not to be considered as reflecting the policies of the Navy Department.

1. This study was suggested to us by Commander Marion B. Sulzberger (MC), U.S.N.R., who supplied clinical material and statistics.

In this paper we shall report the results of our investigation of the microbiologic flora of otitis externa and present related clinical observations.

MATERIAL

Patients.—Fifty young white American marines and sailors provided material for study. These patients constituted an unselected group: Each presented himself at sick call with a complaint of "ear trouble" or "ear fungus" and was found on otoscopic examination to have otitis externa. All had been in the tropics for from eight to thirty months. Marines temporarily stationed at the Transient Center, Marianas Area, Fleet Marine Force, for evacuation to the United States made up most of the group.[2] Twenty of the 50 patients had bilateral otitis externa, and 32 had unilateral involvement, thereby providing 72 ears for study.

As a control, the same cultural technics were applied in a study of the microbiologic flora of the external auditory canals of 16 normal ears, representing 8 men. These men had been stationed in the tropics for approximately the same length of time as the patients with otitis externa.

Mediums.—Agar plates streaked for isolation of single colonies were used throughout. Six mediums were employed.[3] These were: (1) Bacto–Sabouraud maltose agar, p_H 7.2; (2) Bacto–Sabouraud maltose agar adjusted to p_H 5.4 by adding 0.4 cc. of normal hydrochloric acid to 100 cc. of agar; (3) Bacto–Sabouraud maltose agar adjusted to p_H 10 by adding 18.2 cc. of normal sodium hydroxide to 100 cc. of agar; (4) Bacto–Sabouraud maltose agar, p_H 7.2, to which 0.015 per cent potassium tellurite was added; (5) Bacto–Sabouraud maltose agar, p_H 7.2, to which 0.05 per cent copper sulfate was added; (6) Bacto–corn meal agar; (7) human blood agar plates prepared by adding 5 per cent defibrinated human blood to agar prepared from Bacto–blood agar base.

The Sabouraud mediums were cooled to 56 C. before the additions of cupric sulfate and potassium tellurite or the adjustments of p_H were made. The agar for blood agar plates was cooled to 42 to 44 C. before the blood was added.

The mediums described were selected in an attempt to provide solid mediums that were optimal for the growth and isolation of fungi. Differences in hydrogen ion concentration were utilized to enhance the growth of widely different fungi: p_H 5.4 for fungi such as Aspergillus and Penicillium; p_H 7.2 for yeasts and the causative agents of deep mycoses; p_H 10 for the pathogenic dermatophytes. Cupric sulfate and potassium tellurite were incorporated in Sabouraud's agar so as to inhibit or prevent the growth of certain bacteria. Bacto-corn meal agar was prepared with a view to its being used in the study and identification of yeasts. Human blood agar, on the other hand, was used so as to learn what bacteria were present, for this medium supports growth of most bacteria whether they are pathogens, normal flora or extraneous saprophytes.

METHOD

Each patient who presented himself with a chief complaint of "ear trouble" or "ear fungus" was subjected to an interview and an examination. The questionnaire

---

2. At the time this study was carried out, marines in large numbers were awaiting evacuation on the point system. Of 1,453 marines who presented themselves as patients for treatment for all causes, 235, or 16.6 per cent, had otitis externa, confirmed by otoscopic examination.

and outline for examination as mimeographed for the examiner will be reproduced in this article.

When otoscopic examination confirmed the history and the chief complaint, material for culture was obtained. The technic employed was to visualize the site of maximal involvement by means of an otoscope, a speculum and artificial light and then bring a small sterile absorbent cotton swab into firm contact with that area. If exudate was present, the swab was withdrawn when saturated; if a fungous colony or a localized area of inflammation was visualized, the swab was rolled over the area and withdrawn carefully. In each instance the swab was immediately transferred to 0.1 cc. of buffered 0.85 per cent sodium chloride solu-

Questionnaire on Otitis Externa: Information Obtained from Each Patient at Time of First Visit

..... U.S.M.C(R) ....
Name (last) First Middle Rate Serial No. Age

....
Organization Type of work

....
List areas where you have had ear trouble Other areas stationed in

Total time since first ear trouble ..... No. of episodes ..... Length of each ..... Length of this episode.....

Symptoms: Check as MILD (MI), MODERATE (MO), SEVERE (SE)
Pain..... Soreness..... in jaw..... Itch.....
Discharge..... Color..... Odor.....
Difficulty in hearing..... Dizziness..... Ringing.....

Check treatment received: (H) if it helped (NH) if it did not
Penicillin..... Sulfonamide compounds..... Cresatin.....
Alcohol..... "Drops"..... Glycerin..... Ointments (type if known)..... Other.....
Treatment of this episode.....
When was last treatment.....

Do you swim often..... Last time..... Do you pick your ears.....
Which side of your head do you sleep on.....

REMARKS:

PHYSICAL: Ear involved..... Involved in past.....
ANATOMY: Length..... Diameter..... Tortuosity.....
CANAL: Swelling..... Redness..... Discharge.....
.....
DRUM: .....
.....
REMARKS:

tion. The saline solution taken up by the swab made it possible to inoculate heavily each of six plates. Isolation of single colonies was effected (1) by merely touching the swab to the human blood agar plate and streaking therefrom by platinum loop, (2) by streaking twice with the swab plain Sabouraud's medium and Sabouraud's medium to which cupric sulfate or potassium tellurite had been added, with further streaking therefrom by platinum loop, and (3) by using the

3. Dehydrated culture mediums (Difco products) were employed in the preparation of all lots of mediums. For details as to the constituents and preparation of each medium, see Manual of Dehydrated Culture Media and Reagents, ed. 7. Detroit, Mich., Difco Laboratories, 1943.

swab only for streaking Sabouraud's medium of p_H 5.4 and of p_H 10. The cultures were incubated at 37 C. for twenty-four hours and thereafter at room temperature for ten days. Each plate was examined daily until all previously identified colonies had been studied. Bacteria and yeasts were identified as to genus and species; the filamentous fungi, as to genus.

RESULTS

The results of the cultural studies are presented first so as to relate them subsequently to the clinical findings.

Microbiologic Flora in Normal Ears.—In any attempt to assess, or to exclude, the possible causal relationship of a micro-organism recovered from an exudate of an ear showing otitis externa, the microbiologic flora of normal ears in the same environment must be known. Accordingly, the technic outlined for the investigation of the microbiologic flora of ears with overt disease was applied to the external auditory canal of each of 16 normal ears.

From the results presented in table 1, it was apparent that the micro-organisms recovered from normal ears in the tropics were those accepted as constituting normal flora of the skin lining the external auditory canal.[4] Cultural studies of normal ears for purposes of con-

TABLE 1.—*Micro-Organisms Recovered from the External Auditory Canals of Sixteen Normal Ears*

Incidence of Recovery Expressed in Terms of Number of Ears from Which Each Organism Was Recovered	Micro-Organisms Recovered from External Auditory Canal Identified as					
	Staphylococcus					
	Aureus	Albus	Micrococci	Sarcinas	Diphtheroids	Bacilli
Total number	1	10	13	8	10	2
Percental number	6	62	81	50	62	12

trol were therefore limited to but 16 ears. It can be seen that the incidence of recovery of each organism is given in an absolute and a percental number. Even though percental numbers based on so few subjects mean little, they are presented to give a rough estimate of the relative incidence of each organism.

THE MICROBIOLOGIC FLORA IN OTITIS EXTERNA

Cultures of exudates from 72 ears with clinical otitis externa, representing 50 patients, yielded 186 strains of micro-organisms that fell into 16 categories, as shown in table 2.

From one to six readily separable organisms were recovered from each ear. It must be recognized that a complete representation of distribution probably is not given in table 2, since colonies belonging

4. Topley, W. W. C., and Wilson, G. S.: The Principles of Bacteriology and Immunity, ed. 2, Baltimore, Williams & Wilkins Company, 1936, pp. 1551-1552.

to other genera undoubtedly were obscured by heavy growths, more particularly growths of Pseudomonas, Proteus, Klebsiella, Actinomyces and Aspergillus.

The incidence of recovery of each organism (based on the number of ears found to yield that organism) is presented in table 2. Sixteen different genera were represented: 3 genera of fungi (Eumycetes) and 13 genera of bacteria (Schizomycetes). Allocation of 2 of the fungi and 4 of the bacteria was based on morphologic studies, and therefore only the genus is given. Separation and identification of the remaining micro-organisms were based on morphologic studies and on biochemical tests. As none of the 29 strains of Corynebacterium was found to belong to pathogenic species, all were lumped as diphtheroids. However, the morphologic similarity of 14 of the 29 strains

TABLE 2.—*Generic Allocation of One Hundred and Eighty-Six Strains of Micro-Organisms Recovered by Single Cultures of Exudates from Seventy-Two Ears with Otitis Externa, Representing Fifty Patients*

	Incidence of Recovery			
	From 37 Left Ears	From 35 Right Ears	Total Number of Ears	Percental Number
Actinomyces	5	3	8	11
Aspergillus	4	4	8	11
Monilia tropicalis	1	1	2	3
Phytomonas	1	1	2	3
Pseudomonas aeruginosa	25	23	48	66
Klebsiella ozaenae	3	3	6	8
Proteus	3	5	8	11
Staphylococcus aureus haemolyticus	6	7	13	18
Staphylococcus aureus nonhaemolyticus	13	4	17	24
Staphylococcus albus	5	4	9	12
Staphylococcus citreus	1	0	1	1
Micrococci	15	7	22	30
Sarcinas	8	7	15	21
Diphtheroids	16	13	29	40
Alcaligenes faecalis	1	0	1	1
Bacilli	1	1	2	3

to Corynebacterium diphtheriae led us to test each of them for toxin production by inoculating them intradermally in normal and in passively immunized guinea pigs. None of the strains showed evidence of toxigenicity. Table 2 also gives absolute and percental numbers for the recovery of each organism [5] and shows that infection occurred in the right and in the left ear with essentially the same frequency.

When each of the 16 micro-organisms listed in table 2 was considered as a possible causative agent of otitis externa, 6 immediately were excluded by reason of falling within the group accepted as normal flora for the skin of the external auditory canal. These 6 were the diphtheroids, the micrococci, the sarcinas and all staphylococci except Staphylococcus aureus haemolyticus. Also excluded

5. Synonyms for Pseudomonas aeruginosa are Pseudomonas pyocyanea, Bacterium pyocyaneum and Bacillus pyocyaneus.

from further consideration were the gram-positive spore-forming bacilli, for they undoubtedly were contaminants, and Alcaligenes faecalis. The latter organism was dismissed as evidence of feces to finger to ear transmission, for it was recovered from a single ear of a habitual ear picker. Numerous colonies of Aspergillus and hemolytic staphylococci were present in the same cultures.

Table 3 lists 3 fungi and 5 bacteria which could on circumstantial evidence be accepted as causative agents of otitis externa. All are generally regarded as limited in pathogenicity. If the significance of the demonstrated presence of single micro-organisms is to be assessed, therefore, it is desirable that one have all data on the incidence of recovery of each organism, both as it occurs individually and as it

TABLE 3.—*Potentially Pathogenic Types of Micro-Organisms Recovered from Seventy-Two Ears with Otitis Externa*

Micro-Organism Recovered	Total Number of Strains Isolated by Single Culture of Each of 70 Ears	Incidence of Recovery When									
				It Was Coexistent with							
		It Was Only Organism Present	Bacteria That Constitute Normal Flora Are Excluded	Actinomyces	Aspergillus	Monilia	Phytomonas	Pseudomonas Aeruginosa	Klebsiella Ozaenae	Proteus	Staphylococcus Aureus Haemolyticus
Actinomyces	8	0	1	..	1	0	0	3	1	2	0
Aspergillus	8	0	3	1	..	1	0	2	0	0	1
Monilia tropicalis	2	0	0	0	1	..	0	1	0	0	0
Phytomonas	2	0	2	0	0	0	..	0	0	0	0
Pseudomonas aeruginosa	48	6	27	3	2	1	0	..	4	6	5
Klebsiella ozaenae	6	0	2	1	0	0	0	4	..	2	0
Proteus	8	0	1	2	0	0	0	6	2	..	0
Staphylococcus aureus haemolyticus	14	0	11	0	1	0	0	5	0	0	..

is coexistently present with other micro-organisms of similar latent pathogenicity, and that these findings be considered in their relationship to the clinical symptoms. Accordingly, table 3 presents data for each of the 8 micro-organisms based on aural source to show incidence of recovery. These data show the incidence (1) when colonies of only a single micro-organism were demonstrated, (2) when bacteria that constitute normal flora were excluded and (3) when the organism was found to coexist with one or more other pathogens. It can be seen that the microbiologic flora commonly contained not one but several organisms with about the same latent pathogenicity. When a micro-organism was present in pure culture, or as the only pathogen among otherwise normal flora, it was assumed that the causative agent for that case had been isolated. On the other hand, the recovery of several organisms from a single case made even a tentative assumption

impossible except as judgment was influenced by the clinical symptoms and the character of the exudate.

Fungi were recovered from only 18 of the 72 ears, as indicated in table 3. When fungi were present, however, they grew profusely with a sufficient number of colonies on each of at least five plates to exclude the possibility of their being incidental contaminants. Moreover, 7 of these 18 ears showed on otoscopic examination macroscopic colonies of fungi in the external auditory canal, and these yielded the following fungi on culture: Aspergillus in 5 cases and Actinomyces in 2. No fungous colonies were seen in cultures of material from 11 ears, but in many of these ears exudate, cerumen and debris were present to obscure the field. Thus in 4 the canal was filled by the greenish purulent exudate of a concomitant infection by Ps. aeruginosa. A fungus was present with Ps. aeruginosa in 6 cases: Actinomyces in 2 and Aspergillus in 4. The only evidence in this study to suggest fungi as etiologic agents to the exclusion of bacteria was the fact that Actinomyces and Aspergillus were recovered from one ear and Aspergillus and Monilia from another in the absence of any other pathogens. The recovery of Monilia tropicalis was limited to 2 ears, in one of which it coexisted with Aspergillus and in the other of which it was present with Pseudomonas.

Ps. aeruginosa occurred commonly and usually was present in severe disease. It was readily recognized in most instances by the effuse, fluorescent and slightly pigmented colonies and by the associated trimethylamine-like odor. Sabouraud's agar containing potassium tellurite was especially effective in bringing out colonies of Pseudomonas. The wide variety of pigmentary changes associated with the growth of strains of this organism ranging from yellow through green to blue-black, led to biochemical tests of each strain. All were Ps. aeruginosa. This was isolated from exudates of two thirds of the infected ears, 48 of 72. It was present in pure culture in 6 ears, as the only organism exclusive of normal flora in 27 and in mixed culture with one or more other possible pathogens in 15 ears.

Klebsiella ozaenae was isolated from the exudates of 6 ears, representing 4 patients. It was the only pathogen recovered from 2 ears, occurred in mixed flora with Ps. aeruginosa in 3 and was present with Proteus in 2. A single ear yielded Klebsiella granulomatis in mixed flora with Ps. aeruginosa, Proteus and Actinomyces. The exudate associated with K. ozaenae was small in amount and of a glairy, mucoid consistency. When Klebsiella was present in association with Pseudomonas, the exudate was greenish.

Staph. aureus haemolyticus was recovered from 14 ears, representing as many patients. When organisms belonging to the normal flora were excluded it remained as the only pathogen in 11 of 14 cases. It was

found in association with Ps. aeruginosa in 4 ears and with both Ps. aeruginosa and Actinomyces in 1 ear.

The demonstration of Phytomonas in otitis externa came as a surprise to us. It was the only organism exclusive of normal flora in exudate from both ears of a single patient. The suggestive causal relationship was strengthened by a distinctive, adhesive, mucoid consistency and a sweet odor of the exudate.

Proteus was recovered from 8 ears, representing 5 patients, but in 7 of the ears it was present in mixed flora. The associated organisms were Ps. aeruginosa in 4 ears, Ps. aeruginosa and Klebsiella in 1 ear, Ps. aeruginosa, Klebsiella and Actinomyces in 1 and Actinomyces in 1. As 4 of the 5 patients who yielded Proteus were habitual ear pickers, it is highly probable that fecal contamination via the index finger was responsible for the presence of the organism. In 3 of the cases of Proteus infection the thin, gray purulent exudate had a typically foul, proteolytic odor.

CLINICAL OBSERVATIONS

The distribution of the clinical manifestations of otitis externa based on the study of 72 ears with otitis externa are summarized in table 4. Study of this table makes apparent a composite clinical picture of otitis externa, but the variation from ear to ear was such that any or all of the manifestations were present in single cases. In no instance was the condition asymptomatic.

Pain, soreness and a feeling of fulness were the most common complaints. From one to all three of these symptoms were present in

TABLE 4.—*Distribution of the Clinical Manifestations of Otitis Externa, Based on a Study of Seventy-Two Ears*

	Ears with Given Local Manifestation								
	Pain	Soreness	Pruritus	Exudate	Feeling of Fulness	Impairment of Hearing	Deafness	Tinnitus	Vertigo
Total number.....	59	51	27	34	51	34	6	5	4
Percental number.....	82	71	38	47	71	47	8	7	6

each case. Other common complaints were pruritus, from slight to moderate impairment of hearing and an aural discharge. The aural exudate usually became apparent on pressure or cleaning but occasionally was sufficiently profuse to escape to the pillow. The extent of deafness, tinnitus and vertigo, recorded as being present in 7 per cent, was indefinite and hard to evaluate.

The anatomic and pathologic observations are summarized in table 5. On otoscopic examination each ear presented evidence of acute or chronic inflammation of the external auditory canal. The process was

usually limited to the inner or the middle third; in some ears, however, it extended inward to the epithelium overlying the tympanum or outward to appear in the concha. Obvious redness, localized or generalized, was seen in all infected ears. Edema of soft tissues and visible exudate were present in about three fourths of the group.

An attempt was made to relate the anatomic configuration of the infected external auditory canal to the presence, the incidence and the clinical manifestations of otitis externa. Accordingly, on otoscopic examination observations were made of the length, the tortuosity and the diameter of each canal. The results, as summarized in table 5, make it apparent that the anatomic findings were within the expected variation for any group of 50 persons. Thus, in three fourths of the patients under study the auditory canal was of average or medium

TABLE 5.—*Summary of Results of Otoscopic Examination of Seventy-Two Ears with Otitis Externa*

	Ears Showing Given Anatomic Finding					Ears Showing Given Pathologic Finding		
	Increased Tortuosity	Small Diameter	Length of External Auditory Canal					
			Long	Medium	Short	Erythema	Edema	Exudate
Total number.....	8	5	12	56	4	72	56	56
Percental number....	11	7	17	78	6	100	78	78

TABLE 6.—*Length of Time Since Onset of First Attack of Otitis Externa*

	Cases	Average Number of Episodes
From 0 to 7 days.....	4	1
8 to 14 days.....	4	1
2 to 8 weeks.....	2	1.5
2 to 6 months.....	17	4
7 to 12 months.....	8	6
1 to 3 years.....	13	6
More than 3 years.....	2	21

length, in 17 per cent it was longer and in 6 per cent it was shorter than average. Other findings were equally inconclusive, for the diameter was less than normal in only 7 per cent and an increase of tortuosity was shown in 11 per cent. It was concluded, therefore, that the anatomic configuration was a negligible factor in determining the presence of otitis externa.

Table 6 summarizes for all 50 cases the duration of disease. Again wide variation among cases was shown as to the duration of a single episode and as to the total number of episodes. In some cases the disease became apparent suddenly, to last a few days and disappear. In others it was characterized by chronicity with exacerbations. In certain cases the history suggests that an inapparent infection persisted with survival of the causative organisms during symptom-free intervals.

EPIDEMIOLOGIC OBSERVATIONS

Epidemiologic studies of these 50 cases were limited to learning whenever possible whether the onset, the clinical manifestations and the microbiologic flora of otitis externa were influenced by habitual or occasional swimming or by ear picking and whether the ear involved was infected because it was the ear slept on.

Only 10 of the 50 patients gave a history of having been in swimming within two weeks before the visit for treatment. Two swam regularly, and 8 occasionally. Several attributed the onset of their illness to swimming; others claimed that their symptoms were aggravated by salt water. However, these opinions undoubtedly were colored by the belief prevalent among military personnel that "ear fungus" results from salt water.

Picking, or insertion of a finger, usually the index finger, into the external auditory meatus, was common, being indulged in by 50 per cent of the group (23 of 50). Of these 23, 8 were "lifetime" habitual pickers, 3 were "lifetime" occasional pickers and 12 picked at their ears only when having ear trouble.

It was thought that the increase of warmth and humidity in the canal of an ear habitually slept on might result in that ear being the one to show infection. No evidence was obtained to support this conjecture.

SUMMARY AND COMMENT

The microbiologic flora of otitis externa is made up of a variety of micro-organisms. By reason of ready recoverability, predominance in numbers and associated pathologic manifestations, the following micro-organisms, singly or in association with other bacteria or fungi, were accepted as causative agents: Ps. aeruginosa, Staph. aureus haemolyticus, K. ozaenae, Phytomonas (sp. n.d.), Aspergillus and possibly Proteus (sp. n.d.), M. tropicalis and Actinomyces. Worthy of comment are the somewhat equivocal results of the present investigation so far as they bear on the role of fungi in otitis externa. Because fungi generally are accepted as the primary causative agents of this disease, while bacteria are regarded as secondary invaders or opportunists of no significance, we fully expected, by the utilization of a variety of mediums known to be optimal for the isolation and growth of pathogenic fungi, to isolate a fungus from each ear. The problem was undertaken so as to determine what fungi act as causative agents and not to learn if they were present. It was a surprise, therefore, when only 25 per cent of the infected ears (18 of 72) yielded fungi and to find that invariably the fungus recovered was but one of several pathogens in a mixed flora. Moreover, all strains of fungi fell into 3 genera recognized as being of slight pathogenicity: Aspergillus, Actinomyces and Monilia. Visualization on otoscopic examination of

fungous colonies in ears with otitis externa, and recovery from such ears of aspergilli, even though coexistent with at least one other fungus or bacterium, made it apparent in the present series of cases that fungi do have a role in the causation of this disease. On the other hand, failure to recover fungi in 75 per cent of the cases under study certainly means that fungi play no part in many, if not in most, cases of otitis externa. It was concluded, therefore, that a fungus can be one of the primary infectious agents in otitis externa but that recovery of a fungus in a case of otitis externa is to be regarded in the same light as the isolation of certain bacteria.

Otitis externa, as exemplified by the present series of cases, was characterized from patient to patient by from slight to marked variation in the symptoms, objective physical findings and microbiologic flora. Nevertheless, when considered in aggregate, the cases roughly fell into four groups: Group A was made up of cases with subjective symptoms that usually included pain, soreness, a sense of fulness and often pruritus; they were also characterized by moderate to marked edema of the soft parts, even to involvement of the tragus and the preauricular tissues; by a yellowish to gray purulent exudate, and by the presence, in the purulent exudate of Ps. aeruginosa in pure culture, associated with normal flora or associated with other bacteria and fungi. Group B was characterized by pain, sharp or aching, and a feeling of fulness with little in the way of soreness, edema or other symptoms; by localized erythema, slight edema and rarely a macroscopic pustule on otoscopic examination, and by presence of Staph. aureus haemolyticus as the predominant organism in the flora. Group C consisted of cases in which there were subjective symptoms of fulness and slight impairment of hearing, with otoscopic examination disclosing macroscopic colonies of fungi subsequently shown to belong in the genus Aspergillus or Actinomyces. Group D was made up of an ill defined group of cases in which for the most part the patient had, as symptoms, pain, itching and a sense of fulness of the ear; the pathologic observations included erythema, edema and exudate, variable but usually extensive, and the biologic flora included several pathogens. Findings associated with certain bacteria, such as Klebsiella, Phytomonas and Proteus, have been described in the foregoing pages. In a case from which hemolytic staphylococci and Ps. aeruginosa were isolated, both ears were covered from the external meatus to the scapha with a crusted, weeping, eczematoid dermatitis that exuded a thin seropurulent fluid. The exudate that welled from the external auditory meatus, on the other hand, was purulent and greenish yellow. Both types of exudate contained staphylococci and Ps. pyocyanea.

It is probable that the first attack of this disease represents an entirely fortuitous set of circumstances. As the external auditory canal

is open to the exterior, micro-organisms gain entrance to it by being on forcibly introduced fingers or foreign objects employed in "cleaning" the ear or are brought into the canal incidentally with air or water as the vehicle. Once beyond the meatus, they are in a moist chamber that contains in the normally present cerumen and cellular detritus an adequate culture medium to support the growth of many fungi and bacteria. Any inflammation, however mild it may be, will yield products to support further growth. Thus, the mere presence of infection is not surprising. What is of interest is that the normal flora exists as harmless commensals and that pathogens common to systemic diseases are not encountered. The micro-organisms present in otitis externa constitute an intermediate group, neither contaminants nor unequivocal pathogens: Ps. aeruginosa, K. ozaenae, Staph. aureus haemolyticus, Aspergillus, Actinomyces, Monilia tropicalis, and others. Perhaps it is more than coincidence, for the very invasiveness that characterizes certain bacteria may be a reason for their absence. Contrariwise, a total inability to establish themselves may characterize other micro-organisms, such as the penicillia. Thus penicillia predominate in the flora of air and almost anything exposed to the humidity and the heat of Guam, yet not one of them was isolated on culture from any case of otitis externa.

The role of otitis externa as a cause of partial to complete disability of military personnel is little recognized except by members of the medical corps. Common among men stationed in tropical or subtropical climes, it is characterized by exacerbations and recurrent attacks that result in time lost for daily treatment at sick call, a loss in general efficiency from impairment of hearing, and chronic foci of infection. In not a few cases it is intractable to treatment, with the result that the patient is evacuated to the United States.

CONCLUSIONS

1. Otitis externa is a clinical entity with a high morbidity of military personnel on tropical duty.

2. The microbiologic flora of the external auditory canal in otitis externa, which in most cases is mixed, differs from the flora of the normal auditory canal in the same clime by the inclusion of one or more pathogens.

3. The causative agent of otitis externa can be any one of a variety of bacteria and fungi. Among these are Ps. aeruginosa, K. ozaenae, Staph. aureus haemolyticus, Aspergillus and probably Monilia tropicalis, Actinomyces and Proteus.

4. The role of fungi in otitis externa has undoubtedly been overemphasized, whether as primary or secondary agents of infection.

5. As the response to treatment in otitis externa is determined by the causative agent present, exudate from each ear should be cultured before treatment is started.

6. The efficacy of each of the medicinal preparations and antibiotic drugs recommended for treatment of otitis externa should be tested by employing groups of 20 cases in which the etiologic factors are known.

University of Rochester School of Medicine and Dentistry, Rochester, N. Y. (J. T. Syverton).

Hospital of the Rockefeller Institute, New York (W. R. Hess).

A STUDY OF CHRONIC SUPPURATIVE OTITIS MEDIA AT THE PENNSYLVANIA SCHOOL FOR THE DEAF

WILLIAM J. HITSCHLER, M.D.
AND
MARY FRANCES VASTINE, M.D.
PHILADELPHIA

THIS study of chronic suppurative otitis media at the Pennsylvania School for the Deaf includes the causes, the physical findings, the degree and the kind of deafness, various forms of nonoperative treatment and their value, and the relationship of nasal, bacterial and roentgen findings to the disease.

The children reside at the school, where there are ample facilities for examination and treatment and where the living conditions of all are similar. One of us (M. F. V.) also lived at the school.

Each patient was seen and treated almost daily. The appearance of the ears, the amount of discharge, the nasal condition and other data were recorded, and treatment was administered. Most of the children had been at the school for several years. Hence there were available records of their past health and treatment. Because of these special conditions, though the number of cases is small, the observations made and the conclusions reached may be of value.

All related tests and examinations were carried out. They included bacterial studies, roentgen studies of the mastoid process and the sinuses and audiometric examinations.

The daily observations began in January 1942 and ended in June of that year when the children left to spend the summer at their homes. Follow-up examinations were made on their return in October.

Sixteen children, 3 per cent of the entire student body of 533, had chronic suppurative otitis media at the beginning of the study. Of the 16, 10 had bilateral and 6 had unilateral involvement; hence a total of 26 ears were studied.

The ages ranged from 8 to 17 years, with 14 years being the average. Ten were boys, and 6 were girls. In the whole school the ratio of boys to girls is approximately 8 to 6. Thirteen of the 16 children had had previous treatment at the school, some for a number of years.

The histories were obtained from the children, from the school records and from separate communications received in conference with the parents. The age at which the otitis media began ranged from 6

months to 8 years, with 4 years as the average. In 7 cases the aural involvement dated definitely to scarlet fever; in 3 cases, to measles. In 5 other instances the infection probably followed measles, although, owing to deficiencies in the histories obtainable, this was not a certainty. In 1 case the condition was said to have been brought on by a cold.

The predominance of scarlet fever and measles as causative of chronic otitis media makes one wonder if the thoughts presented by Kopetzky [1] and Shambaugh Jr.[2] are not correct. These men surmised that in many cases chronic otitis media begins as such and is not just a prolongation of acute otitis media. On the other hand, Dr. Horace J. Williams [3] stated that 98 per cent of the children in the Municipal Hospital for Contagious Disease in Philadelphia who have otitis media in connection with scarlet fever leave that institution with dry ears.

The duration of the infection varied from six to fourteen years. The average duration was nine years. This may seem too long a time for the infection to have been present to expect a cure from conservative treatment. However, Lederman [4] said, "The duration of the suppurative disease plays no deciding role in advising heroic measures." In other words, conservative treatment may be just as efficacious in chronic otitis media of long duration as in that of short duration.

There was a history of bilateral otitis media in every case. However, only 26 ears were included in this study, since the other 6 were dry and have remained so since. In 4 cases the discharge was constant, while in 10 it was intermittent. Four children had undergone simple mastoidectomy, 2 bilaterally, apparently without benefit. Fourteen of the children had had their tonsils and adenoids removed. Curiously enough, the only children who had not been treated by tonsillectomy and adenoidectomy were the very ones on whom bilateral mastoidectomy had been performed. Comparing the age of the onset of the otitis media with the age at which the tonsillectomy and the adenoidectomy were done, we found that in nearly every instance these operations were performed after the onset of the otitis media and also after the onset of deafness. Comparing the age of onset of the otitis media with the age of onset of deafness, the two were found to be the same in all but 2

1. Kopetzky, S. J.: Chronic Mastoiditis and Its Therapy, J. Iowa M. Soc. **26**:665-670 (Dec.) 1936.

2. Shambaugh, G. E., Jr.: Pathology and Treatment of Chronic Suppurative Otitis Media, cited by Boies, L. R.: Management of Ear Diseases in Children, Graduate Course in Otolaryngology, American Academy of Ophthalmology and Otolaryngology, 1942.

3. Williams, H. J.: Personal communication to the authors.

4. Lederman, M. D.: Twenty Years' Experience with Iodine Powder (Sulzberger) in Conservative Treatment of Aural and Nasal Suppuration, Laryngoscope **47**:110-125 (Feb.) 1937.

cases; in one of these the deafness came first, and in the other the otitis media came first. However, the histories as to time of onset of deafness did not seem definite and were therefore probably not reliable.

Of the 16 audiograms made to determine the threshold of hearing for air conduction in these patients, 4 showed hearing losses which were greater for low tones, 6 the flat type of curve, and 5 showed losses which were greater for the higher tones. In 1 child there was not enough residual hearing to be recorded by the audiometer. In all but 2 cases the result of the Rinne test was negative. In 1 case the result was positive, and in 1 air conduction equaled bone conduction.

The losses of hearing for air conduction are shown in table 1.

TABLE 1.—*Losses of Hearing for Air Conduction in Children with Chronic Suppurative Otitis Media*

Loss in Decibels *	Ears
Less than 40	2
40 to 49	4
50 to 59	6
60 to 69	6
70 and more	8

* The loss is the average of the losses for 512, 1024 and 2048 double vibrations per second.

The 6 ears that were dry at the beginning of the study and have remained so showed practically the same range of hearing when the study was completed as when it was begun.

It is interesting to compare the data for these children who had chronic suppurative otitis media with those obtained previously by Dr. Hughson [5] for the whole school. He found that (1) 54 per cent were said to have been born deaf, (2) most of the children had greater loss for high tones, (3) bone conduction was better than air conduction, (4) the hearing loss was great. In the first quartile it ranged from 60 decibels at 128 double vibrations to 90 decibels at 4096.

One sees then that those who were born deaf are not included in this group with chronic suppurative otitis media. Their deafness is probably from another cause. This group with chronic suppurative otitis media as a whole showed more of a conductive type of audiometric curve, although several of the ears in this group had the greatest loss in the high tones (as did the whole school). Bone conduction was better than air conduction in both groups. The hearing loss was less in the group with chronic suppurative otitis media.

5. Hughson, W.; Ciocco, A., and Palmer, C.: Studies of Pupils of Pennsylvania School for the Deaf: Auditory Acuity, Arch. Otolaryng. **29**:403-416 (March) 1939.

The question arises as to how much of the loss of hearing was caused by the chronic suppurative otitis media. It is to be noted that in our series the difference between the loss in the group whose ears were running and the loss in the group whose ears were not running is slight. However, it must be remembered that each child had a history of both ears discharging at some time in his life. The loss of hearing seems to be too great in many instances to be caused by the otitis media alone. Chronic otitis media in itself does not cause more than 50 to 60 decibels' loss at the maximum. Usually the loss is much less than this. The conclusion is, therefore, that at least some of these children have a lesion in addition to that caused by the chronic otitis media. The histories seem to indicate that it is not a congenital one.

Could there be some sclerosis at the foot of the stapes or is there a lesion of the organ of Corti itself of such a nature that vibration conducted through cochlear fluid will not register on the eighth nerve while vibration conducted through bone will? Theoretically, such a lesion could be located in the movable parts of the organ of Corti. In some cases a perceptive lesion might be the principal cause of the deafness, particularly in those cases in which the ears are not discharging. However, if this premise is true, the negative result of the Rinne test is difficult to explain. Hughson [5] said that "the 'biologic constitution' is a factor in the causation of the deafness," something that occurs "in the uterine life or in the elements transmitted to the child by his parents." Perhaps, in spite of the negative histories, this is the cause of deafness in some of these cases.

Is the incidence of chronic otitis media in an institution like the Pennsylvania School for the Deaf higher or lower than that in the general population? This study showed it to be 3 per cent. According to a recent report,[6] 266,226 children in the public schools of Philadelphia showed an incidence of 0.29 per cent. This means that the incidence of chronic suppurative otitis media is approximately ten times higher in the Pennsylvania School for the Deaf than in the Philadelphia public schools. The difference is not so great if children with some degree of deafness are compared. Among 2,709 of the public school children in New York who have 9 decibels or more of hearing loss, there were 290, or slightly over 1 per cent, who had chronic suppurative otitis media. In the New York day school for the deaf there were 24 with purulent otitis among 400 children examined, an incidence of 0.6 per cent.[7] "Purulent otitis" probably includes the acute as well as the chronic form.

6. Statistical Report of the Division of Medical Inspection of Public Schools, Philadelphia, 1938.

(Footnotes continued on next page)

The discharge of 13 ears of 11 children was submitted to bacteriologic examination. Culture showed a mixed infection. It was unsatisfactory in 1 case. On smears, gram-positive cocci were found in 7 cases, spirilla in 3, gram-negative bacilli in 3 and Bacillus pyocyaneus in 1. Others [4] also have found that there is usually a mixed infection. Special examinations were made for fungi in every instance, but none were found. No sheets of desquamated epithelium and no cholesterol crystals were seen. The absence of the latter, however, is not significant, because the acetone used in the staining process (Grams) would have dissolved them. No efforts were made to obtain specimens from the attic or from the antrum of the mastoid process as suggested by Morrison.[8]

Aural examinations made before instituting treatment showed the membrana tympani to be entirely absent in 14 ears. However, in 4 of these the short process of the malleus was visible. In 5 ears the posterior part of the membrane alone remained. In 2 ears the membrane was laid back against the promontory. In these the membrane had a central defect through which granulation tissue protruded. Only a small amount of Shrapnell's membrane remained in 2 ears, and in 1 of these the short process was present. The 3 remaining ears presented only a small rim of drum membrane.

Most writers [9] on chronic suppurative otitis media have classified their cases into at least two groups: (1) those without involvement of bone, distinguished by a central perforation only, and (2) those with involvement of bone, having marginal or attic perforation. All agree that in the first group cure is simple, but many assert that in the second group only indifferent success is observed with conservative treatment. Despite the fact that in several of these cases the drum membrane was back against the promontory and showed a central perforation and in some only a rim or other part of the membrane remained, we classify them all as being of the second, or more serious, group. We do this because in every case there had probably been some destruction of the ossicular chain and in no case was there any remnant of the drum

7. Acoustically Handicapped Children, Report of Subcommittee on Acoustically Handicapped Children of Committee for Study of the Care and Education of Physically Handicapped Children in Public Schools of the City of New York, Board of Education, New York, 1941.

8. Morrison, W.: Modern Conception of Pathology, Diagnosis and Treatment of Chronic Suppurative Otitis Media, Ann. Otol., Rhin. & Laryng. **51**:5-21 (Feb.) 1942.

9. (*a*) Ashley, R. E.: Surgical and Nonsurgical Care of the Chronically Discharging Middle Ear, Arch. Otolaryng. **33**:993-1003 (June) 1941. (*b*) Shambaugh, G. E., Jr.: End Results in Treatment of Chronic Suppurative Otitis Media, Illinois M. J. **75**:180-183 (Feb.) 1939.

membrane that could be considered as even approximating the normal in appearance or structure. The membranes that were not against the promontory had changed to thick fibrous tissue.

Granulation tissue was present in 14 tympanic cavities. It varied from areas pinpoint in size to areas 2 mm. in diameter. No polyp formation was seen. Five middle ears were partially or completely dermatized. In 8 there was mucous membrane only. In 3 of these the mucosa appeared slightly swollen, while in 5 the mucosa was smooth and normal in color.

The discharge was profuse in 4 ears, while it was moderate in amount in 13. The discharge obtained from 7 ears was slight and that from 2 was very slight. In 19 ears the discharge was foul smelling. It was moderately foul in 4, and only a faint odor was detected in 3. There was no demonstrable relationship between the types of bacteria found in cultures and the degrees of odor.

Roentgenograms of the mastoid areas were obtained in all of the 16 cases. Some increased density, i. e., sclerosis, was present in each of the 32 mastoid bones. In the group under discussion (26 mastoid processes) the density was complete, with no cells showing in 7 films. In 7 others the sclerosis was less marked, but still no cellular outline was seen. In 13 a moderate increase in density was shown, together with poor cell formation in other parts of the mastoid bone. In 2 there was an area of apparent rarefaction posterior to the antral region. Radiologists differed in opinion as to whether or not this was cholesteatoma. No evidence of abscess formation was reported.

Comparing the duration of the discharge with the degree of sclerosis revealed no significant data. In fact, there was less sclerosis in those ears in which the duration of the discharge was longer. There was no relationship between improvement with treatment and the amount of sclerosis present. Just as many moderately sclerosed as completely sclerosed ears were included in the improved and in the unimproved subgroup.

We conclude that the roentgenographic studies of this group have not added materially to our information except in a negative way; i. e., they eliminated abscess formation. Ashley [9a] said that roentgenograms are of little value in diagnosis because sclerosis is so frequently present.

Nasal examination of the 16 patients showed the nasal structure to be normal in 12. There was septal deviation in 4. There was no demonstrable relationship between septal deviation and persistence of aural discharge. The nasal mucosa appeared normal in 5 children. It was reddened to a greater or lesser extent in 11. Nine children had pus in the nose and 2 an excess of mucus. Transillumination indicated clouding of one or more sinuses in 4 patients. In each of the 4 one

antrum was denser than the other, while involvement of 2 frontal sinuses was indicated by this examination. The findings by transillumination and those by roentgenography often did not agree. One maxillary or one frontal sinus often showed more clouding by transillumination but less involvement by roentgenography, or vice versa. The roentgenologist reported clouding in 3 frontal sinuses and 4 sphenoid sinuses, with possible exudate in 1 of the sphenoid sinuses. There was clouding of the ethmoid in every child, although it was slight in 6 cells. One or both maxillary sinuses appeared pathologic in 13 of the children. In 6 of these the antral findings were such as to indicate the probable presence of exudate. In 5 of these 6 the antrums were irrigated, and exudate was found in 3. It must be noted that there was at least a six week period between the taking of films of the sinus and the antral irrigations.

Nasopharyngoscopic examinations showed that the nasopharynx was reddened in 13, normal in color in 1 and pale in 2 children. Pus was seen in 3 and excess mucus in 3. One or both eustachian tubes were reddened in 8 and swollen in appearance in 4. Pus was seen issuing from a tube in 1.

Adenoids were present in the nasopharynx in 12 of the 16 children. In 2 the adenoids did not encroach on the tubes. In 5 they touched the posterior lips of the tubes. The tubes were partially closed by adenoidal tissue in 2 children. In 3 others there was complete closure of one or both tubes due to overgrowth of this tissue. Of the 14 children who had undergone tonsillectomy and adenoidectomy, no adenoidal tissue was present in 3, a small amount was present in 5, a moderate amount in 3 and a large amount in 3. It may be seen that the percentage of children in whom there was considerable regrowth of adenoidal tissue after adenoidectomy was high. There was a great deal of adenoidal tissue present in the 2 children who had not had their tonsils and adenoids removed. In 1 of these, so excessive was the growth that neither eustachian tube could be visualized.

In 7 children the throat appeared nonpathologic, while in 9 it was slightly reddened.

The nonoperative treatment of chronic suppurative otitis media has been discussed by many. Shambaugh Jr. [9b] has had 23 cures in 36 cases in which there was secondary cholesteatoma, using attic irrigations. He expressed the belief that in such cases the chronic suppurative otitis media often has resulted from scarlet fever and measles occurring in childhood and that the tympanic membrane has a large perforation, including the pars tensa and the pars flaccida, with foul discharge. He mentioned another group of cases, in which again the chronic otitis media often has been caused by scarlet fever and measles, with somewhat

similar findings in the ear, that require operation. Tobey [10] pointed out that nasal and pharyngeal disease should be corrected, but mentioned a fact which was noted also in this study—that septal deformities do not affect the ear. He expressed the belief that local therapy is successful in from 70 to 80 per cent of the cases. His treatment consists of syringing the middle ear and the attic, burning out granulations, removing cholesteatoma, drying, and instilling boric acid and alcohol. Lederman [4] has had good results, even in the most severe cases of otitis media, by using boric acid–iodine powder.[11] Tremble [12] favored the use of urea. However, all agreed with Hill [13] when he said, "Perhaps the greatest single virtue of any form of treatment is in the mechanical cleansing . . . ." It may bear repeating that in the cases herein considered the ear was cleansed (if discharging) almost daily for five months.

In the endeavor to estimate the value of several forms of local therapy of chronic otitis media, the following agents were employed: powdered sulfathiazole, powdered sulfanilamide and microcrystalline sulfadiazine powder.[14] In addition, zinc ionization and dry wipes were used. All aural granulation tissue was cauterized, and nasal treatment was administered in those cases in which otorrhea persisted despite intensive local therapy and in which there was marked evidence of sinal involvement.

In evaluating the amounts of discharge of the ears we employed eight gradations. These were "dry," "moist," "very slight," "slight," "slight to moderate," "moderate," "moderate to profuse" and "profuse." In each case the ear was thoroughly dry wiped before local medication was employed. This wiping consisted of a thorough cleansing by means of a cotton applicator saturated with a solution of boric acid and alcohol, after which the canal and the fundus were dried with plain sterile cotton. Prior to this study most of the children had been treated at the school by competent otologists. The treatment consisted of dry wiping, instillation of boric acid–iodine powder, baking with infra-red rays and the usual nasal therapy. It was given once or twice a week, or less frequently, depending on the condition of the child's ears at the time.

10. Tobey, G. L., Jr.: Chronic Suppurative Otitis Media, Tr. Am. Otol. Soc. **20**:140-149, 1930.

11. Boric acid–iodine powder is prepared by mixing an iodine solution of desired strength with dry, finely powdered boric acid and evaporating the solvent. The result is an intimate association of iodine and boric acid, in which the iodine is the active agent.

12. Tremble, G. E.: Use of Urea and Caroid in Chronic Otitis Media, Canad. M. A. J. **40**:149-153 (Feb.) 1939.

13. Hill, F. T.: Chronic Discharging Ear, Dis. Eye, Ear, Nose & Throat **1**:112-116 (April) 1941.

14. Microform sulfadiazine powder was supplied by the Smith, Kline & French Laboratories.

Powdered sulfathiazole was the first agent used. Twenty-three ears received an average of sixteen treatments each over a three week period (table 2). At the end of this period, 18 showed improvement, 11 of which became dry. Four ears showed no change. One ear was worse. However, every one of those that became dry had a recurrence of the discharge at some time. Four of these 11 ears were free of discharge from two to three months, 1 was dry for one month, while in the other 6 the discharge recurred within ten days or less. It is worthy of note that 1 ear which remained dry for eleven weeks after this treatment had

TABLE 2.—*Results of Treatment with Sulfathiazole Powder*

	Amount of Discharge				Total Number of Treatments	Previous Treatment by Us	
Ear	When Treatment Started	One Week Later	Two Weeks Later	Three Weeks Later			Result
1	Profuse	Moderate	Moderate	Slight to moderate	15	None	Improved
2	Moderate	Slight to moderate	Moist	Dry	18	None	Dry
3	Moderate	Slight	Dry	Dry	17	None	Dry
4	Moderate	Moist	Moist	Moist	16	None	Improved
5	Moderate	Slight to moderate	Moist	Dry	12	None	Dry
6	Slight	Moist	Slight	Moist	14	None	Improved
7	Slight	Moist	Slight to moderate	Moderate	14	None	Worse
8	Slight	Moderate	Moderate	Slight	17	None	Unchanged
9	Slight	Slight	Moist	Moist	17	None	Improved
10	Slight to moderate	Slight	Moist	Dry	16	None	Dry
11	Slight	Profuse	Moderate	Slight	16	None	Unchanged
12	Very slight	Moist	Dry	Dry	17	None	Dry
13	Very slight	Moist	Dry	Dry	17	None	Dry
14	Moderate	Moist	Slight	Slight	17	None	Improved
15	Moderate	Moist	Moderate	Slight	17	None	Improved
16	Moderate	Moderate	Moderate	Moderate	17	None	Unchanged
17	Profuse	Profuse	Profuse	Profuse	17	None	Unchanged
18	Profuse	Slight	Moist	Moderate	17	None	Improved
19	Profuse	Very slight	Dry	Dry	16	None	Dry
20	Slight	Dry	Slight	Dry	15	None	Dry
21	Moderate	Dry	Moderate	Dry	15	None	Dry
22	Slight	Moist	Slight	Dry	16	None	Dry
23	Slight	Moist	Dry	Dry	17	None	Dry
24							
25							
26							

never been free of discharge since the time of admission to this institution seven years before, and 1 which remained dry for ten weeks had never remained so for longer than four weeks. The 9 other ears which became dry with sulfathiazole had been free of discharge at least once before.

Sulfanilamide powder was next employed (table 3). It was instilled approximately thirteen times over a four week period in each of 16 ears. The ears in which it was used were those which had not become dry with the sulfathiazole or in which there had been a recurrence of discharge. Three ears (nos. 24, 25 and 26) received sulfanilamide instead of sulfathiazole as their initial local treatment. These averaged eight treatments over a two week period.

Of these 16 ears that had received sulfathiazole and were now given sulfanilamide, 4 showed improvement, 1 of which became dry. Six remained the same, and 2 became worse. The 4 others, which had had a recurrence of the discharge, became dry again with the sulfanilamide, as they had with the sulfathiazole. There was improvement

TABLE 3.—*Results of Treatment with Sulfanilamide Powder*

	Amount of Discharge							
Ear	When Treatment Started	One Week Later	Two Weeks Later	Three Weeks Later	Four Weeks Later	Total Number of Treatments	Previous Treatment by Us	Result
1	Slight	Moderate	Very slight	Slight to moderate	Slight	13	Sulfathiazole	Unchanged
2								
3	Dry	Dry	Slight	Dry	.....	3	Sulfathiazole	Dry after recurrence
4	Moist	Moderate	Moist	Dry	Dry	11	Sulfathiazole	Dry
5								
6	Moist	Dry	Moist	Moist	Moderate	9	Sulfathiazole	Worse
7	Moderate	Slight	Slight to moderate	Slight	Moderate	9	Sulfathiazole	Unchanged
8	Profuse	Slight	.....	Moderate	Slight	11	Sulfathiazole	Improved
9	Moist	Moist	.....	Moist	Slight	11	Sulfathiazole	Worse
10								
11	Moderate	Slight	Slight	Moderate	Moderate	12	Sulfathiazole	Unchanged
12								
13								
14	Slight	Slight to moderate	Moist	Slight to moderate	Slight	14	Sulfathiazole	Unchanged
15	Moderate	Moist	Slight to moderate	Moist	Slight to moderate	14	Sulfathiazole	Unchanged
16	Moderate	Slight	Slight to moderate	Moderate	Moderate	14	Sulfathiazole	Unchanged
17	Profuse	Slight	Moist	Profuse	Slight	14	Sulfathiazole	Improved
18	Moderate	Moist	Moist	Profuse	Moist	14	Sulfathiazole	Improved
19								
20								
21	Dry	Slight	Dry	Slight	Dry	13	Sulfathiazole	Dry after recurrence
22	Dry	Slight	Dry	Slight	Dry	9	Sulfathiazole	Dry after recurrence
23	Dry	Slight	.....	Moist	.....	9	Sulfathiazole	Dry after recurrence
24	Moderate to profuse	Slight to moderate	Slight	Slight to moderate	.....	10	None for 7 weeks	Improved
25	Moderate	Slight	Dry	.....	.....	7	None	Dry
26	Profuse	Slight	Slight to moderate	.....	.....	7	None	Improved

in all of the 3 ears in which sulfanilamide was given as the initial treatment. One of these became dry. Only 1 ear that had not become dry with the sulfathiazole became so with the sulfanilamide. However, 1 ear (no. 4), in which the discharge recurred after sulfathiazole therapy had been discontinued and which was treated with sulfanilamide, remained dry for the duration of the study.

Zinc ionization treatment was next employed. Hett and co-workers[15] reported extensive use of ionization. They used it only when all other therapy had failed. They stated that it was not effective if the bone was necrosed and that the ear remained dry only as long as

TABLE 4.—*Results of Ionization Treatment*

	Amount of Discharge							
Ear	When Treatment Started	One Week Later	Two Weeks Later	Three Weeks Later	Four Weeks Later	Total Number of Treatments	Previous Treatment by Us	Result
1	Slight to moderate	Slight to moderate	Moderate to profuse	.....	.....	1	(1) Sulfathiazole (2) Sulfanilamide (3) Dry wipes	Unchanged
2								
3								
4								
5								
6	(1) Profuse (2) Moderate	(1) Slight to moderate (2) Profuse	Slight	Profuse	Moderate	2	(1), (2) and (3)	Unchanged
7	(1) Moderate (2) Moderate	(1) Slight to moderate (2) Moderate	Profuse	Profuse	Moderate	2	(1), (2) and (3)	Unchanged
8	Slight to moderate	Moderate to Profuse	Moderate to Profuse	Moderate to Profuse	Moderate to Profuse	1 1	(1), (2) and (3) (1), (2) and (3)	Unchanged Unchanged
9								
10								
11	(1) Moderate (2) Profuse	(1) Moderate to profuse (2) Moderate	(1) Moderate (2) Moderate to profuse	.....	.....	2	(1), (2) and (3)	Unchanged
12	(1) Slight to moderate	(1) Slight to moderate	Slight	.....	.....	1	(1), (2) and (3)	Unchanged
13								
14								
15								
16	Moderate	Profuse	Moderate	Moderate to profuse	.....	1	(1), (2) and (3)	Unchanged
17	Profuse	Profuse	Profuse	Profuse	.....	1	(1), (2) and (3)	Unchanged
19								
20								
21								
22								
23								
24								
25								
26								

it would with any other treatment, i. e., one or two months or more. In 2,652 cases they obtained 2,058 cures—a rate of 78 per cent. The

15. Hett, G. S.; Wells, A. G., and Levick, G. M.: Ionization in Cases of Suppuration of the Middle Ear, Proc. Roy. Soc. Med. (Sect. Otol.) **22**:139-146, 1929.

current used was 0.2 to 0.1 milliampere. Levick [16] obtained cures in 60 to 70 per cent of patients in hospital clinics and more in private practice. Friel [17] reported that in cases in which the otorrhea was due only to tympanic sepsis the results of ionization were immediate, complete and consistent. Schmidt [18] cured aural discharge in well over 80 per cent of patients by ionization. MacFarlan [19] had success in "many simple cases of chronically discharging ears" and expressed the conviction that often "the discharge will cease with this method when other methods have failed."

TABLE 5.—*Results of Treatment with Dry Wipes*

	Amount of Discharge							
Ear	When Treatment Started	One Week Later	Two Weeks Later	Three Weeks Later	Four Weeks Later	Total Number of Treatments	Previous Treatment by Us	Result
1	Slight to moderate	Slight to moderate	Slight	.....	.....	13	Sulfathiazole Sulfanilamide	Improved
2								
3								
4								
5								
6	Profuse	Slight	Profuse	.....	Profuse	16	Sulfathiazole Sulfanilamide	Unchanged
7	Moderate	Moderate to profuse	Profuse	.....	Moderate	16	Sulfathiazole Sulfanilamide	Unchanged
8	Slight to moderate	Moderate to profuse	Moderate to profuse	Moderate to profuse	Moderate	19	Sulfathiazole Sulfanilamide	Worse
9	Moist	Dry	Slight	Slight	Slight	9	Sulfathiazole Sulfanilamide	Unchanged
10								
11	Slight	Moderate to profuse	Moderate	Moderate	Moderate to profuse	18	Sulfathiazole Sulfanilamide	Worse
12	Moderate	Slight to moderate	Slight	.....	.....	7	Sulfathiazole Sulfanilamide	Improved
13								
14	Slight	Slight	Slight to moderate	.....	.....	12	Sulfathiazole Sulfanilamide	Unchanged
15	Very slight	Slight	Moderate	.....	.....	12	Sulfathiazole Sulfanilamide	Worse
16	Moderate	Profuse	Profuse	.....	.....	8	Sulfathiazole Sulfanilamide	Worse
17	Profuse	Profuse	Profuse	.....	.....	12	Sulfathiazole Sulfanilamide	Unchanged
18	Slight	Profuse	Profuse	.....	.....	12	Sulfathiazole Sulfanilamide	Worse

In ionization a direct current is passed into the ear (anode pole), which has been previously cleansed and filled with a 2 per cent solution

16. Levick, G. M.: Ionization in Cases of Suppuration of the Middle Ear, J. Laryng. & Otol. **45**:53-59 (Jan.) 1930.

17. Friel, A. R.: Zinc Ionization and Zinc Electrolysis in Diseases of Throat, Nose and Ear, Dublin J. M. Sc., February 1922, pp. 640-648.

18. Schmidt, W. H.: Personal communication to the authors.

19. MacFarlan, D.: Ionization: Circuit Plans for Inexpensive Unit, Arch. Otolaryng. **21**:456-458 (April) 1935.

of zinc sulfate. The cathode is placed on an arm of the patient. The current is slowly raised to from 2.5 to 6 milliamperes, depending on the patient's toleration. The treatment is continued from three to five minutes, and then the current is slowly turned off. This is the technic of MacFarlan.[19] We used it in 9 of those ears which after treatment with both sulfathiazole and sulfanilamide exhibited discharge which was slight to moderate or greater in amount. Ionization was repeated in 4 of the 10 ears. The ears were dry wiped during this time. No improvement was noted in any ear.

Twelve ears were treated with dry wipes for a two to four week period (table 5). These 12 ears received an average of thirteen such treatments. Improvement in amount of discharge was noted in 2 ears. There was no change in 5 ears, and the discharge became more profuse in 5 ears as a result of this treatment.

The efficacy of microcrystalline sulfadiazine powder was next assayed (table 6). The powder was used in 16 ears. There were approximately seventeen treatments for each ear over a four week interval. Six were improved, 3 of which became dry. Eight ears showed no change, and in 2 ears the discharge became more profuse. Of the 3 dry ears, 2 had been dry before, both with the first therapeutic agent used, i. e., sulfathiazole. The 1 ear which became dry for the first time with sulfadiazine remained so for two weeks.

There were granulations in 16 of the 26 ears when this study was begun and in 7 ears at the end of it. Under treatment with the sulfonamide drugs and without cauterization, the granulations disappeared in 10 ears. As for the discharge in theses 10 ears, it ceased in 7 (only temporarily in 1) and remained the same in 3 ears. In 8 ears treated locally with sulfonamide drugs granulations either became larger, remained the same or appeared where before there had been none. The discharge remained the same in 6 of these 8 ears and decreased in amount in the other 2. Cauterization with a 50 per cent solution of silver nitrate was done in 5 ears in which granulations either persisted or appeared for the first time after intensive local therapy. The granulations disappeared from 1 ear. In 3 ears they became smaller. In 1 they remained the same size. The amount of discharge remained unchanged in 4 of these and lessened in 1. Two other ears were cauterized at the same time that local therapy was first instituted; granulations disappeared in the one ear and remained the same size in the other. The discharge from both improved. In general, the granulations varied with the amount of discharge, i. e., less with less discharge and more with more discharge. These granulations appeared to be secondary rather than primary. In other words, effective treatment reduced the granulations and the discharge, while cauterization

without effective treatment reduced neither the granulations nor the discharge.

Systematic treatment of the nose was instituted in 9 cases in which the ear continued to discharge despite other treatment. In 8 cases it consisted of the daily use of packs saturated with a 1 per cent solution of ephedrine in the middle meatus. Daily irrigations with isotonic

TABLE 6.—*Results of Treatment with Microcrystalline Sulfadiazine Powder*

Ear	Amount of Discharge: When Treatment Started	One Week Later	Two Weeks Later	Three Weeks Later	Four Weeks Later	Total Number of Treatments	Previous Treatment by Us	Result
1								
2	Slight	Very slight	Very slight	.....	.....	10	Sulfathiazole	Slightly improved
3								
4								
5								
6	Moderate	Profuse	Profuse	Profuse	.....	25	(1) Sulfathiazole (2) Sulfanilamide (3) Dry wipes (4) Ionization	Unchanged
7	Moderate	Moderate	Moderate to profuse	Moderate to profuse	.....	25	(1), (2), (3) and (4)	Unchanged
8	Moderate	Slight	Moderate to profuse	Moderate	.....	25	(1), (2), (3) and (4)	Unchanged
9	Slight	Dry	Very slight	Slight	Slight to moderate	25	(1), (2), (3) and (4)	Slightly worse
10								
11	Moderate to profuse	Slight	Moderate to profuse	Slight to moderate	Moderate	25	(1), (2), (3) and (4)	Unchanged
12	Very slight	Very slight	Very slight	.....	.....	9	(1), (3) and (4)	Unchanged
13								
14	Moderate	Dry	Slight	Dry	Dry	15	(1), (2) and (3)	Dry
15	Moderate	Slight	Slight	Slight	Dry	18	(1), (2) and (3)	Dry
16	Moderate	Moderate	Moderate	Moderate	Moderate	23	(1), (2), (3) and (4)	Unchanged
17	Profuse	Profuse	Profuse	Profuse	Profuse	18	(1), (2), (3) and (4)	Unchanged
18	Moderate	Slight	Slight	Slight	Moderate	18	(1), (2), (3) and (4)	Unchanged
19								
20								
21								
22								
23	Slight	Very slight	Very slight	Dry	Dry	7	(1), (2)	Dry
24	Slight	Slight	Slight	Moderate	Moderate	19	(2)	Worse
25	Moderate	Moist	Dry	Dry	Slight	11	(2)	Improved
26	Moderate to profuse	Dry	Moist	Moderate	Slight	11	(2), (4)	Improved

solution of sodium chloride were administered in the ninth case. Irrigations of the maxillary sinuses were performed in several cases. The greatest number of nasal treatments given in any case was twenty, and the least number received was seven. In no case did a lessening of the aural discharge accompany this treatment. However, improvement of the nasal condition on the whole was not marked.

There were 26 discharging ears when this study was begun and 20 discharging ears at the end of the school year. The majority of these 20 ears had less discharge than previously.

In an endeavor to find what factors were present in the 6 ears which responded best to treatment, the following analysis was made: The history, including the original cause of the otitis media, and the duration, whether intermittent or continuous, gave no hint. Roentgenograms of the mastoid process showed all stages of sclerosis from slight sclerosis with some cellular elements to very dense sclerosis. Most of the ears showed granulations before the study, none after they became dry. Head colds developed in 3 of the 6 children after the ears were dry. One of these showed no effect of the cold on the ears; the ears of the other 2 discharged again, but only temporarily. Nasopharyngoscopic examination showed no adenoidal tissue in 2, a slight amount in 1, a moderate amount in 1 and a great deal in 2. The adenoidal tissue touched the posterior lip of each eustachian tube in 3 and covered the tubes completely in 1. There was frank pus in the nasopharynx in 3 of the 6 children and frank pus in the anterior nares in 2. Two had deviated septums. All but 1 had reddened nasal mucosa. Hence the condition of the nose or of the nasopharynx gave no clue. Generally speaking, there was much less involvement of sinuses, as shown by the roentgenograms, in those whose ears became dry. Only 1 child had any sinus described as opaque, while most had but moderate or slight involvement. This contrasts with the more marked involvement encountered in those whose ears did not become dry. The only common finding which serves to differentiate these 6 children with dry ears from the others is this relative paucity of sinal involvement as shown by roentgenograms.

The question of radical mastoidectomy arises. Is this justifiable in those cases that are refractory to conservative treatment? There are reasons against such a procedure. They are:

1. The operation involves both psychic trauma and danger to life.
2. Effective after-care of children is difficult.
3. Radical mastoidectomy often does not result in a dry ear, particularly when reason 2 is taken into consideration.
4. Controlled chronic suppuration of the ear, provided the general health is maintained, and provided there are no complications, is not a great handicap.

The children went home from school in June and returned in October. In this time, as far as could be determined, they had no treatment. While there were 8 ears that had profuse or moderate discharge in June, there were 19 in October. There were 12 ears which had slight

discharge or were just moist in June and 3 in October. There were 6 dry ears in June and 3 in October. The total number of ears in October numbered 25 inasmuch as 1 child did not return to school. These figures show that a considerable part of the improvement from the treatment had been lost over this period.

Table 7 shows the results more graphically. For simplicity, ears with profuse and ears with moderate discharge are combined under one heading and those with slight discharge and those that were moist are combined under another.

From this study of treatment the administration of powdered sulfathiazole, that of powdered sulfanilamide and that of microcrystalline sulfadiazine powder would seem to be equally efficacious in the treatment of chronic otitis media and superior to the other forms of treatment these children had. We noted some caking in the external canal with regular crystals of sulfathiazole, but except for this we are not justified in saying that one of these agents is to be preferred to any other. It

TABLE 7.—*Summary of the Results of Treatment*

Time of Observation	Ears with Profuse and Ears with Moderate Discharge	Ears with Slight Discharge and Ears That Were Moist	Ears That Were Dry
Beginning of treatment (January) (but after some other treatment at school)..	17	9	0
End of period of treatment (June).....	8	12	6
After four months without treatment (October)	19	3	3

might be noted that the solubility of the microform sulfadiazine crystals is greater than that of the usual form.

In our hands, ionization and dry wiping were of little or no benefit in the treatment of the chronically discharging ears. In the evaluation of the effectiveness of the various treatments we have taken into account the fact that each successive agent used was at more of a disadvantage than the preceding agent. This is true because the more recent medication was applied only to the ears which had been refractory to previous treatments.

It is obvious that on the whole the effectiveness of these various treatments has not been marked. In fact, the results have been disappointing. Why should this be so? In the first place, it must be remembered that most of the ears had been previously treated systematically by competent otologists without permanent effect. These ears, therefore, do not represent the average run of ears with chronic suppurative otitis media but the refractory ones. Secondly, all the patients not only had disease of the middle ear but probably had disease of the mastoid bone. It is likely that when there is marked necrosis of

bone, no amount of conservative treatment of the ear or improvement of the condition of the sinuses or the nasopharynx will produce a cure. Other possibilities are: (1) failure to wash out or curet attics and remove diseased ossicles, (2) too intensive treatment, (3) persistence of too much sinal and pharyngeal involvement. Nasal and pharyngeal surgical procedures should perhaps have been attempted.

However, 3 ears have remained dry after four months without treatment. The treatment also made some ears dry that were never dry before, and, what may be more significant, enabled many to be transferred, at least temporarily, from the "profuse" and "moderate" group to the "slight" and "moist" group.

SUMMARY AND CONCLUSIONS

Twenty-six children with chronically discharging ears were studied and treated intensively at the Pennsylvania School for the Deaf.

Measles and scarlet fever were the etiologic factors in almost all of these patients.

The hearing loss was greater than would be expected from the chronic suppurative otitis media alone. Bone conduction was better than air conduction in almost all the patients.

Bacteriologic examination showed a mixed growth.

Apparently, there was involvement of bone in every patient.

Roentgenograms of the mastoid processes were of value only in the exclusion of a possible cholesteatoma or abscess.

There was no relationship between deviation of the septum and persistence of otitis media. There was some involvement of sinuses in every patient.

Many of the children had adenoidal regrowth.

Various forms of treatment were instituted and the results noted. The powdered sulfonamide compounds were most efficacious. Six of the 26 ears became temporarily dry, while most of the ears showed a temporary decrease in the amount of the discharge.

Dr. Walter Hughson gave assistance in the preparation of this paper.

8840 Germantown Avenue.

A STUDY OF THE ATTITUDINAL REFLEXES OF MAGNUS AND de KLEIJN IN THALAMIC MAN

HUGH O'NEILL, M.D.
SANTA ANA, CALIF.

The head is righted by labyrinthine, tactile and optical stimuli; the body by proprioceptive and tactile stimuli acting separately upon both the body and the head. The orientation of the head and of the body takes place in relation to gravity, environment and parts of the body, and the integration of every single factor of this complicated function is doubly insured.—*Magnus.*[1]

THE REFLEXES OF MAGNUS AND DE KLEIJN

The vestibular apparatus, the gyroscope of the head (McNally), by the controls exercised by the dynamic equilibratory semicircular canals and the static equilibratory maculas of the utricle, with ocular tactile, muscular and visceral senses, maintains the balance of the body. Quix expressed the belief that increased flexor tone resulted from pressure of the otolithic membranes on the macula, but to Magnus increased extensor tone followed the pulling of the otolithic membranes on the macula. The years 1907 and 1909 are important milestones to neuro-otologists, for in the former year Bárány published his classic text on the physiology of the semicircular canals, while in 1909 the pharmacologist Magnus and the otologist de Kleijn described the tonic cervical and the tonic labyrinthine reflexes which position the neck and the body in standing. They showed that tonic cervical reflexes govern the position of the head and the neck relative to the body, while the labyrinthine reflexes determine the spatial position of the head in relation to gravity.[2]

The postural activities of a decerebrate animal exhibit (1) local static reactions of one limb, (2) segmental static reactions of one segment of the body and (3) general static reactions embracing more than one body segment.[1a] In the first group of these, the local static reactions, a limb becomes a pillar of support for the body because of

1. (*a*) Magnus, R.: Some Results of Studies in the Physiology of Posture, Lancet **2**:531, 1926; (*b*) Experimental Basis for Theories on Vestibular Function, Proc. Roy. Soc. Med. (Sect. Laryng. & Otol.) **17**:11, 1924; (*c*) Animal Posture, Proc. Roy. Soc., London, s. B **98**:339, 1925-1926; (*d*) Körperstellung, Berlin, Julius Springer, 1924. (*e*) Magnus, R., and de Kleijn, A.: Die Abhängigkeit des Tonus der Extremitätenmuskeln von der Kopfstellung, Arch. f. d. ges. Physiol. **145**:455, 1912.

2. Grinker, R. R.: (*a*) Neurology, ed. 2, Springfield, Ill., Charles C Thomas, Publisher, 1937; (*b*) ed. 3, 1943.

proprioceptive reflexes arising from "stretch" of the extensor muscles. Accompanying this conversion of the limb into a supporting pillar (positive supporting reaction) by myotatic stretch reflexes, a co-contraction occurs at the ankle joint of the lower limb, for example, where the usual phenomenon of reciprocal relaxation of flexor muscles (antagonists of the extensors) does not take place. In order to neutralize the stretch reflex to evoke the negative supporting reaction, extreme plantar flexion of the distal part of the limb must be performed. In the second group, segmental static reactions, the reactions occur by reciprocal innervation, with crossed extensor reflexes presenting, and when there is applied to one limb a nociceptive stimulus, causing a flexion response, there occurs in the contralateral limb an immediate increase in extensor tone designed to support the weight of the trunk moving away from the noxious stimulus. In the third group, concerned with more than one segment of the body, the general static reactions include tonic cervical reflexes, observed if and when vestibular control is negated by labyrinthectomy. For example, unilateral movement of the head will cause development of extensor tone on the shoulder side, with that of flexor tone showing contralaterally. Again, if the head is dorsiflexed, the forelimb extends with hindlimb flexion, while in ventriflexion of the head there is forelimb development of flexor tone with hindlimb extension. Finally, if pressure is applied in the region of the seventh cervical vertebra, relaxation of all four limbs occurs. The afferent arc of these reflexes, according to Spiegel,[3a] runs in the first three cervical nerves, with their center in the first and second cervical segments.

Tonic labyrinthine reflexes, on the other hand, which can be studied only after cervical neurectomies (the tonic cervical reflexes being thus ablated) arise from the utricle and the saccule, the static equilibratory organs of the vestibular apparatus. These, being static in nature, are excited by movement only, with resultant response in the vestibular nerve via the macular sensory epithelium, the utricles acting in a conjoined manner and the saccules unilaterally. In human decerebrate rigidity, lacking cervical neurectomies, the head and neck must be immobilized for study of the tonic labyrinthine reflexes. The utricular effect on body tone is bilateral but that on the muscles of the neck is unilateral since in a cervically denervated decerebrate specimen placed on its back, with the snout 45 degrees above the horizontal, the otoliths thus pulling on the macular hair cells, all four extremities are in maximal tone. Contrariwise, if the specimen is on its abdomen with the snout 45 degrees below the horizontal, the otoliths resting on the macular

3. (*a*) Spiegel, E. A.: Experimentelle Neurologie, Berlin, S. Karger, 1928. (*b*) Spiegel, E. A., and Sommer, I.: Neurology of the Eye, Ear, Nose, and Throat, New York, Grune & Stratton, Inc., 1944; (*c*) p. 551.

hair cells, the reverse is true and the tone is minimal. These tonic reflexes from the utricle, then, act on the muscles of both the limbs and the neck, with the difference that the effect is bilateral in the case of the limbs but contralateral in that of the neck. Following unilateral labyrinthectomy, the head will deviate, but no changes in tonus of the limbs will result; the labyrinthine effect on the limbs is both direct and indirect, since the labyrinths by secondarily acting on the muscles of the neck cause tonic cervical reflexes, which then react on the limbs.[2] In the first two years of human life there is a fixed resemblance to animals in respect to the changes in tone produced by the tonic cervical reflexes, and infants up to 6 months of age display labyrinthine reflexes arising from the semicircular canals.[4] The tonic labyrinthine reflexes are centered in the medulla oblongata caudad at the plane of entry of the vestibular nerves.[2] When both labyrinthine and cervical tonic reflexes are present, the effect of changes of posture is the algebraic sum of these reflexes, and combinations clinically puzzling may follow, since if one is fortunate enough to see tonic cervical reflexes in a patient, it is unlikely that the patient will simultaneously duplicate the condition of the cervically denervated decerebrate animals with full play being given to the tonic labyrinthine reflexes. For example, when a decerebrate animal is placed on its back, ventral flexion of the head produces relaxation of all four limbs, as the labyrinth is in the position for minimal tone, but the tonic cervical reflexes cause not only flexor tone in the upper extremities but extensor tone in the lower extremities; the algebraic reflex sum will be forelimb flexion with unchanged position of the hindlimbs.[2] To Magnus these tonic reflexes are attitudinal because they are not only indefatigable but are elicitable so long as the head is in a fixed position, the bodily attitudes changing with changes of the head—the proximal end of the body acting as a leader for the distal end.[1d] These attitudinal reflexes are present at all times but are seen in gross "released" effects in the decerebrate animal, in which the inhibitory mechanisms of encephalization are removed. The effects of the labyrinths on the lower spinal centers are mediated not only through the large-celled lateral nucleus of Deiters, giving rise to the descending vestibulospinal tract of the spinal cord, but through the rubroreticular and the reticulospinal tracts, since by the release of the reticular structures exaggerations of the tonic cervical and labyrinthine reflexes are produced.[2]

Five co-operating groups of righting reflexes were also described by Magnus as follows: The zero condition entails when a bilaterally

4. Schaltenbrand, G.: (*a*) Normal Bewegungs-und Lageraktionen bei Kindern, Deutsche Ztschr. f. Nervenh. **87**:23, 1925; (*b*) The Development of Human Motility and Motor Disturbances, Arch. Neurol. & Psychiat. **20**:720 (Oct.) 1928.

labyrinthectomized thalamus-sectioned animal is held up by the body. The animal can be twisted in any direction without its making an attempt to bring its body or head back into normal position, a state realizable in the classic example of a patient with acquired deaf-mutism, due to destruction of the membranous labyrinths, submerged in water. The patient would drown owing to this zero condition.

1. Labyrinthine righting reflexes: If the labyrinths are intact in a thalamic animal, the head always returns to its normal position if the animal is held free. These reflexes originating in the otolithic maculas

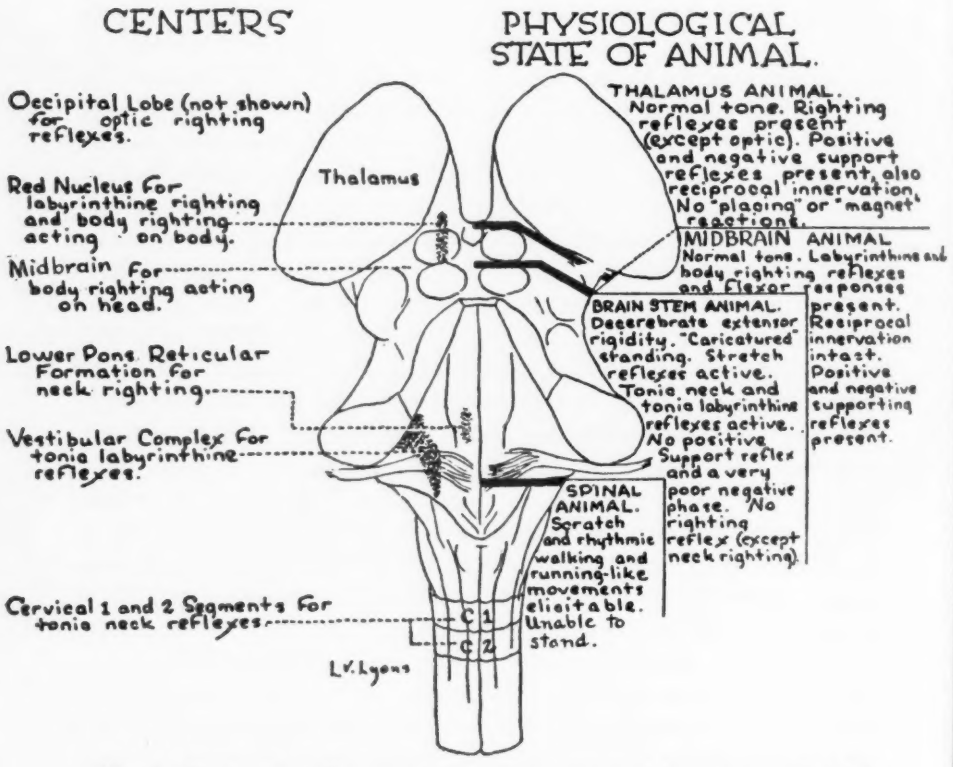
CENTERS
PHYSIOLOGICAL STATE OF ANIMAL.
Occipital Lobe (not shown) for optic righting reflexes.
Red Nucleus for labyrinthine righting and body righting acting on body.
Midbrain for body righting acting on head.
Lower Pons Reticular Formation for neck righting.
Vestibular Complex for tonic labyrinthine reflexes.
Cervical 1 and 2 Segments for tonic neck reflexes.
Thalamus
C 1
C 2
L.V. Lyons
THALAMUS ANIMAL. Normal tone. Righting reflexes present (except optic). Positive and negative support reflexes present, also reciprocal innervation. No "placing" or "magnet" reactions.
MIDBRAIN ANIMAL Normal tone. Labyrinthine and body righting reflexes and flexor responses present. Reciprocal innervation intact. Positive and negative supporting reflexes present.
BRAIN STEM ANIMAL. Decerebrate extensor rigidity. "Caricatured" standing. Stretch reflexes active. Tonic neck and tonic labyrinthine reflexes active. No positive Support reflex and a very poor negative phase. No righting reflex (except neck righting).
SPINAL ANIMAL. Scratch and rhythmic walking and running-like movements elicitable. Unable to stand.

Fig. 1.—Explanatory diagram of the centers concerned in posture and righting and the different physiologic states in animals (mammals) following transections at various levels of the neuraxis. (From Brock.[18])

of the labyrinth act on the muscles of the neck; they are observable in blindfolded infants, who when prone bring the head to normal by dorsiflexion; the symmetric righting reflexes come into play by utricular stimulation, with the opposing saccules keeping the head straight. De Kleijn declared that the function of the saccules is obscure.[3b]

2. Body righting reflexes of tactile, muscular and visceral sense acting on the head: If a labyrinthectomized thalamic animal is placed

with its body on the floor, sense organs of pressure and touch cause the head to rotate to its normal position, while if it is held suspended in the air, deprived of tactile and pressure sense, the head does not move. If while the animal rests on the floor a stimulation equal to that of floor pressure is placed on the upper side of the body, the original reflex is nullified, because the labyrinthine gravitational effect is negated by the body righting reflexes in relation to the floor.

3. Cervical righting reflexes: When, with the head correctly oriented by the labyrinthine and body righting reflexes, the animal realines the thorax with the twisted neck, it does so by virtue of these cervical righting reflexes.

4. Body righting reflexes on the body: These are seen whenever in the preparation the neck may be out of line with the thorax; the body attempts to get into an upright posture in response to superficial stimuli. Schaltenbrand [4b] showed that these reflexes are present in young children and infants until the lower segmental reflex activities are controlled by the myelinization of the higher centers. From his studies of young children he further showed that the Landau reflex appears in the first and second years of childhood; i. e., when the child is held face downward with the hand under its trunk, it reflexly throws up its head and legs, curving the spine concavely. The standing-up movement of man also is a body righting reflex on the body, the child indeed righting itself like any quadruped, rolling over and pushing itself up by its hands (fig. 15). Further, tonic cervical reflexes have been seen in fetuses of 3 to 5 months of age, removed by uterine sectioning.[2]

5. Optic righting reflexes: These enable a cervically denervated labyrinthectomized animal to maintain a normal position, provided its eyes are fixed on some object. Magnus and de Kleijn therefore, by 1912, had shown that the righting reflexes cooperated with the postural and proprioceptive reflexes to determine normal attitudes, and since in the decerebrate animal these reflexes described by Magnus and his school are one leg of the tripod in the establishment of human decerebrate rigidity, the exhibition of such reflexes in a patient who survived the ghastly state of midbrain release is, I believe, of interest to neuro-otologists, since the term "decerebrate" carries the connotation of the terminal state.

DECEREBRATE RIGIDITY

In 1896 experiments were made by Sherrington [5] in a study of the postural reflex in the experimental animal transected at the level of

5. Sherrington, C.: (*a*) Cataleptoid Reflexes in the Monkey, Proc. Roy. Soc., London **60**:411, 1896-1897; (*b*) Decerebrate Rigidity and Reflex Coordination of

(Footnote continued on next page)

the crura cerebri. He observed that an exaggerated type of extensor hypertonus developed, which, even when slight, could be brought out in full by passive flexion and extension of a limb. This condition was described as decerebrate rigidity: "a condition which ensues on removal of the forebrain by transection at any of the levels in the mesencephalon or in the thalamencephalon in its hinder parts." The attitude of the animal, i. e., the erect head and neck, the unyielding back, the erect tail and the uplifted lower jaw, is due to tonus in the muscles, which posturally resist gravity in the erect position, and this posture of the decerebrate animal Sherrington described as reflex standing. The reflex tonus obtains in and is confined to those muscles which maintain the animal in an erect attitude. Reflex tonus is postural contraction; decerebrate rigidity is simply reflex standing.[5] It is commonly acknowledged that the centers for labyrinthine and body righting reflexes lie in the midbrain (Rademaker[6]), the neck righting reflexes in the pons and the optic righting reflexes in the cortex (Spiegel[3b]). The question of cerebellar influence on tone and posture is not clear, since man is no cat, much less a rabbit,[2] and the homologizing of experimental animals of differing phylogenetic strata with man has led to contradictory results. Decerebrate rigidity can be produced in the decerebellated dogs of Rademaker[6] with the righting reflexes unimpaired, and while hypotonia is a feature of cerebellar disease in man, significantly hypertonia occurs in decerebellated dogs. Pollock and Davis[7] expressed the belief that an inhibitory effect is produced on the postural activities of the brain stem by the cerebellum, since decerebellation leads to an increase in tonic labyrinthine reflexes.

A theory of the nervous system taught by Hughlings Jackson[8] postulated a hierarchy of three developmental levels. Jackson taught that as new and higher levels were added to or superimposed on pre-

---

Movements, J. Physiol. **22**:319, 1897-1898; (*c*) The Integrative Action of the Nervous System, New Haven, Conn., Yale University Press, 1906; (*d*) On Plastic Tonus and Proprioceptive Reflexes, Quart. J. Exper. Physiol. **2**:109, 1909; (*e*) The Flexion-Reflex of the Limb, Crossed Extension-Reflex and Reflex Stepping and Standing, J. Physiol. **40**:28, 1910; (*f*) Postural Activity of Muscle and Nerve, Brain **38**:191, 1915; (*g*) Problems of Muscular Receptivity, Nature, London **113**:892 and 929, 1924.

6. Rademaker, G. G.: Das Stehen, Berlin, Julius Springer, 1931.

7. Pollock, L. J., and Davis, L.: (*a*) Studies in Decerebration, Am. J. Physiol. **98**:47, 1931; (*b*) Reflex Activities of a Decerebrate Animal, J. Comp. Neurol. **50**:377, 1930. (*c*) Davis, L. E.: Decerebrate Rigidity in Man, Arch. Neurol. & Psychiat. **13**:569 (May) 1925.

8. Jackson, H.: A Contribution to the Comparative Study of Convulsions, Brain **9**:482, 1886; cited by Grinker[2]; Cerebellar Paralysis with Rigidity, Brain **24**:425, 1906; The Croonian Lectures on the Evolution and Dissolution of the Nervous System, Brit. M. J. **1**:591, 660 and 703, 1884.

existing structures ("encephalization"), old functions were suppressed or discarded as new ones were acquired. As a corollary, when "new" structures have been removed temporarily or permanently by surgical procedure or by disease, there is ablation of the corresponding functions (negative effect), resulting in release of lower, more primitive levels, with production of a grotesque, atavistic carcicaturing of these inhibited "old level" effects (positive effect). Cases of alleged human decerebrate rigidity then should be interpreted in accordance with the hypothesis of Jackson, the concepts of Sherrington and the schema of Magnus and de Kleijn as yardsticks, but in homologizing the state of a mesencephalically transected animal and a clinical state in man it should be kept in mind that when man's shrewlike ancestors took to trees and left quadrupedal locomotion behind, the forelimbs were emancipated to become hands.[9]

In an experimental animal, then, decerebrate rigidity results from the transection of the brain stem at any level between the entering eighth nerves, caudad, and a plane bounded dorsally by the posterior limits of the anterior quadrigeminal bodies and ventrally by the mamillary bodies, cephalad; the supervening state results from an admixture of flexor and extensor overactivity.[5] Clinical decerebrate rigidity, on the other hand, is ill defined, with variously enunciated postulates, and the reported cases are meager in number; they are in dispute, and they embrace for the most part cases of tumor in moribund persons, cases of tuberculous meningitis in children[9a] and cases of softening.[2] In man it should present the following criteria: The antigravity muscles should exhibit resistance to passive elongation; there should be present no modifying postural reactions; the patient should be unable to walk, and the higher central control of the central antigravity mechanism must be abrogated. The rigidity present also should demonstrably be due not only to the stretch reflex in the extensor muscles themselves but also reflexly to the labyrinthine proprioceptors and to the receptors of stretch in the cervical muscles. Importantly (1) there should be absence of specific postural reflexes thus resulting in decerebrate standing, (2) the posturally contracted extensor muscles should be accompanied by a like diminution of tone in the flexor muscles, (3) the animal should lose not only its ability to right itself, but when the body is placed in imbalance, it should topple over, and (4) the limbs should be rigidly extended.

9. Cobb, S.: Review on the Tonus of Skeletal Muscle, Physiol. Rev. **5**:518, 1925; Speech Disorders and Their Treatment, New York, Philosophical Library, Inc., 1943.

9a. Wechsler, I. S.: Tonic Neck Reflexes in Tuberculous Meningitis in Children, J. A. M. A. **85**:510 (Aug. 15) 1925.

Walshe [10a] in studies of the hemiplegic state, after noting the presence of tonic cervical reflexes, stretch reflexes and increased extensor tone, with the work of Sherrington and Magnus and de Kleijn in mind, represented the spasticity of the hemiplegic state as human "hemidecerebrate" rigidity, and an observation by Bazett and Penfield [11] gives some support to Walshe's view. To Kinnier Wilson [12] tonic muscular spasms in extension represent the decerebrate state, and, according to Walshe, this concept is due to Wilson's adoption of a fallacious standard, that of attitude. The standards or criteria enunciated by Walshe [10b] for establishing a diagnosis of decerebrate rigidity in man are: (1) that the rigidity must be the spasticity of a lesion of the pyramidal system, (2) that there must be present the reflexes of Magnus and de Kleijn, with the interruption between tonic and phasic reflexes, and (3) that there must be a constantly falling temperature. The condition will result finally in "the terminal quadripedal decerebrate rigidities" of Cushing.[13a] In 1923 Walshe [10g] reported a case of suprapituitary tumor with, significantly, high intracranial pressure and dilation of the lateral ventricles, with acute secondary hydrocephalus, in a patient with "quasidecerebrate" rigidity; reflex activity was marked, but the arms were flexed and not extended, and later the condition passed to the stage of failing reflex activity and tonic fits. Langworthy [14] denied that the case reported by Walshe was one of decerebrate rigidity; to him it showed an interruption only of cerebropontocerebellar control. Further, in criticism of Walshe's clinical definition, he disagreed with

10. Walshe, F.: (*a*) The Physiological Significance of the Reflex Phenomena in Spastic Paralysis of the Lower Limbs, Brain **37**:269, 1914; (*b*) On the Genesis and Significance of Spasticity and Other Disorders of Motor Innervation, ibid. **42**:1, 1919; (*c*) On Disorders of Movement Resulting from Loss of Postural Tone with Special Reference to Cerebellar Ataxy, ibid. **44**:539, 1922; (*d*) On Certain Tonic or Postural Reflexes in Hemiplegia with Special Reference to the So-Called "Associated Movements," ibid. **46**:1, 1923; (*e*) Observations on the Nature of Muscular Rigidity of Paralysis Agitans and on Its Relationship to Tremor, ibid. **47**:159, 1924; (*f*) On the Syndrome of the Premotor Cortex (Fulton) and the Definition of the Terms "Premotor" and "Motor," with a Consideration of Jackson's Views on Cortical Representation of Movements, ibid. **58**:49, 1935; (*g*) Decerebrate Rigidity of Sherrington in Man, Arch. Neurol. & Psychiat. **10**:1 (July) 1923; (*h*) A Case of Complete Decerebrate Rigidity in Man, Lancet **2**:644, 1923.

11. Bazett, H. C., and Penfield, W. G.: A Study of the Sherrington Decerebrated Animal in the Chronic as Well as Acute Conditions, Brain **45**:185, 1922.

12. Wilson, S. A. K.: On Decerebrate Rigidity in Man and the Occurrence of Tonic Fits, Brain **43**:220, 1920.

13. Cushing, H.: (*a*) Intracranial Tumors, Springfield, Ill., Charles C Thomas, Publisher, 1932; (*b*) p. 2; (*c*) p. 102.

14. Langworthy, O. E.: The Control of Tonus After Injuries to the Brain and the Spinal Cord, Medicine **11**:225, 1932.

Walshe's assumption that in the erect posture the forelimbs were freed from postural control, an assumption which had enabled Walshe to circumvent facilely the obvious anomaly of a decerebrate animal upper extremity extensor tonus and human so-called "hemidecerebrate" upper extremity flexor tonus. Fulton [15f] curtly dismissed the cases of Walshe [10g, h] as instances of decorticate and not of decerebrate rigidity. Fulton [15g] likewise described Davis' case [7c] (suprasellar cyst with decalcification in a child), reported from Cushing's clinic in 1925, as exhibiting decorticate posture with tonic cervical reflexes and lengthening and shortening reactions. According to Fulton, the case was one of decorticate and not of decerebrate rigidity. Decorticate rigidity occurs when the ablation of cerebral tissue is limited to the most recently acquired part of the cerebrum, its mantle or cortex; the deficiencies are far less than those produced by mesencephalic transection (bulbospinal animal) (Bard [16]).

Brain [17] also denied Walshe's hypothesis that the pyramidally controlled prehensile forelimb function is released by abrogation of the pyramidal tract. He theorized that the flexor tonic posture is a modification of the extensor evoked by the assumption of man's erect attitude, quadrupedal stance being to Brain an anthropoidal posture of the forelimbs, with erectness causing a repatterning of a fixed attitude,[18] in short, the emancipation of the forelimb. His explanation is substantiated in the case reported in this paper. Twenty-one years before Walshe [10g] reported the case in question, Cushing,[13b] in 1902, in operating for a suspected intracranial tumor, encountered a huge internal hydro-

15. (*a*) Fulton, J. F.: Muscular Contractions of the Reflex Control of Movement, Baltimore, Williams & Wilkins Company, 1926. (*b*) Fulton, J.; Liddell, E., and Rioch, D.: The Influence of Experimental Lesions of the Spinal Cord upon the Knee Jerk: I. Acute Lesions, Brain **53**:311, 1930. (*c*) Fulton, J., and Liddell, E.: The Influence of Unilateral Destruction of the Vestibular Nuclei upon Posture and the Knee Jerk, ibid. **53**:327, 1930. (*d*) Fulton, J.; Jacobsen, C. F., and Kennard, M. A.: A Note Concerning the Relation of the Frontal Lobes to Posture and Forced Grasping in Monkeys, ibid. **55**:524, 1932. (*e*) Fulton, J. F., and Bailey, P.: Tumors in the Region of the Third Ventricle: Their Diagnosis and Relation to Pathological Sleep, J. Nerv. & Ment. Dis. **60**:1, 1929. (*f*) Fulton, J. F.: Physiology of the Nervous System, New York, Oxford University Press, 1938, p. 164; (*g*) p. 165.

16. Bard, P.: A Diencephalic Mechanism for the Expression of Rage, with Special Reference to the Sympathetic Nervous System, Am. J. Physiol. **84**:490, 1928. Macleod, J. J. R.: Physiology in Modern Medicine, ed. 8, St. Louis, C. V. Mosby Company, 1938, p. 115.

17. Brain, W. R.: On the Significance of the Flexor Posture of the Upper Limb in Hemiplegia, with an Account of a Quadrupedal Extensor Reflex, Brain **50**:113, 1927.

18. Brock, S.: The Basis of Clinical Neurology, Baltimore, William Wood & Company, 1938. Brain.[17]

cephalus. Antecedently he had noted quadripedal rigidities due to the presence of a tumor, disclosed at autopsy, designated by Welch as an interpeduncular teratoma of the pituitary body anlage. He mentioned the rigidities again thirty years later [13c] in describing the case of a 2 month old infant who died of an intracranial teratoma with "acquired decerebrate rigidities." To him decerebrate rigidity had the finality of the terminal state.[13a]

In 1930 Manson and Ferguson,[19] reporting a case which they had observed for a decade, a case of "terminal decerebrate rigidity" with extension of all four extremities, in which at autopsy there was found an endothelioma of the right prerolandic area, with softening in the midbrain and degeneration of pyramidal tracts, concluded that the results of complete single release, i. e., Walshe's hemiplegic state, are not identical with those of complete double release, in which in the decerebrate state both cortical and striatal efferent systems are affected.

Jackson [8] in 1906 described full extensor tonus in cerebellar lesions, and Wilson [12] in 1920 expressed the belief that involuntary choreiform and athetotic movements were part of decerebrate attitudes. Brock and Wechsler [20] wrote that postural defects seen in extrapyramidal disease are also human examples of partial decerebrate rigidity. Wilson,[12] analyzing human postures in terms of animal decerebrate rigidity, described (1) cases of tonic fits with decerebrate attitudes, (2) cases of tonic fits with decerebrate rigidity and (3) cases of decerebrate rigidity without tonic fits. He included both tonic fits and extrapyramidal syndromes, as did Brock and Wechsler.[20] He insisted on the occurrence of partial manifestations of decerebrate rigidity in man, for example, the overpronation of the held-out hand (figs. 10 and 12) and the equinovarus position of the foot (figs. 3, 10, 12 and 15), which obviously are segmental static reactions of Magnus and part, with tonic cervical reflexes, of the threefold mechanism in the production of decerebrate rigidity. Wilson did not study the cervical and labyrinthine reflexes in his reported cases.[15f] In 1931 Davis and Pollock,[7b] reporting a case of alleged antemortem decerebrate rigidity, stated that although the complete experimental lesion would probably never be observed in the clinic, yet the essentials of that lesion may be observed, i. e., interference with voluntary motor power and plastic rigidity able to be offset by phasic spinal reflexes. The reflexes of Magnus and de Kleijn, they asserted, incorrectly I believe, are not necessary to establish the decerebrate state although they agreed that their presence in the absence

19. Manson, J. S., and Ferguson, F. R.: Decerebrate Rigidity in Man, Brit. M. J. **2**:767, 1930.

20. Brock, S., and Wechsler, I. S.: Loss of the Righting Reflex in Man, Arch. Neurol. & Psychiat. **17**:12 (Jan.) 1927.

of postural reflexes adds strong evidence for the existence of such a condition. They expressed the belief that gradual loss of thermic control without irregularities of pulse and respiration must be shown, and they, as did Walshe, discarded most of Wilson's cases, denying that tonic fits were observed in decerebrate rigidity, and made the point that attitudes should not be criteria for judging decerebrate rigidity because strychnine poisoning and meningitis produce a similar (but not a decerebrate) condition. On the other hand, Brock [18] stated that the closest approach to the decerebrate state in man to him is seen in children with meningitis. Wechsler [9a] also asserted that the tonic cervical reflexes of Magnus and de Kleijn are essentially a sign of decerebrate rigidity and that this sign occurred in 50 per cent of children with tuberculous meningitis observed by him, the meningitic and encephalitic process being preponderantly interpeduncular at the midbrain.

SPASTICITY AND RELEASE PHENOMENA

The question of cerebral influence on postural tone is important. With obvious diverse degrees of operative mutilation, the results in different experimental animals are not in agreement. Thiele in 1905 expressed the opinion that the inhibiting influences were thalamic and not cortical. Warner and Olmsted [21] in 1923 reported contralateral extensor hypertonus following ablation of one cerebral cortex anterior to the optic thalamus. Langworthy,[14] summarizing the animal experimental data, stated that the cerebral cortex inhibits and controls the postural reflex but that it is uncertain whether the hypertonus which is a sequel of cerebral injuries is due to primary motor neuron overaction or removal of inhibitory control acting through the cerebellum, while Magnus,[1] on the other hand, denied that the cerebellum plays any part.[3b] Fulton,[15d, f] on the basis of ablation experiments in monkeys, expressed the belief that lesions in area 4 of Brodmann produce motor paralysis and flaccidity and that for the production of spasticity the premotor area must be injured. He expressed the belief that, by an action on the lower motor centers, tonus is inhibited by the premotor area, ablation of which results in spasticity by releasing decerebrate rigidity.[18] Walshe,[10f] denying Fulton's assumption that the premotor cortex is the cortical representative of the extrapyramidal motor system, contended that the more extensive the destroying lesion of the cortex in the region immediately anterior to the fissure of Rolando the more severe is the hemiplegia. To Fulton [15f] the phenomenon of spasticity is an extrapyramidal release of cortical origin.

21. Warner, W. P., and Olmsted, J. M. D.: Influence of Cerebrum and Cerebellum on Extensor Rigidity, Brain **46**:189, 1923.

Foerster [22] ascribed to pyramidal motor cortex destruction "negative symptoms," such as the loss of the specialized motor innervations which control and modify the extrapyramidal synergies, and positive symptoms, consisting of the release of extrapyramidal synergies. Hines,[23] working on rhesus monkeys, showed that removal of "area four" and sectioning of the pyramids produced paralysis, with loss of isolated voluntary movements approaching the loss suffered by man after lesion of his motor cortex. The removal of the pyramidal system alone leaves the extrapyramidal system in full activity, with the result that isolated voluntary movements of discrete parts are lost, together with marked decrement in normal tone of all muscles. Hines therefore classified the extrapyramidal systems as those which initiate movements and those which inhibit both movement and tonic states.

In insulin shock, as Frostig [24] has shown, the syndromes actually present indicate both the degree of progression of the insulin effect and the magnitude of the suppressed area of the brain, with the symptoms progresing in an order inverse to the phylogenetic age of the layers. With cortical suppression, syndromes referable to the basal ganglions are released, followed by midbrain release and finally release of the centers in the medulla.

CEREBRAL SUBDURAL HEMATOMA

The production of the quadripedal "decerebrate" clinical state displaying the reflexes of Magnus and de Kleijn is explicable in the case reported in this paper on the basis of (1) compression of the temporal lobe with abrogation of the corticospinal and extrapyramidal tracts and (2) "dislocation" of the midbrain and contralateral supratentorial compression of the brain stem, caused when a subdural cerebrospinal "hydroma" pushed the cerebellum into the foramen magnum. Munro,[25b] reporting 310 verified cases of cerebral subdural hematoma, showed that the solid type of hematoma described by Putnam and Cushing in 1935 is the least common of all subdural hematomas which come to operation.

---

22. Foerster, O.: The Motor Cortex in Man in the Light of Hughlings Jackson's Observations, Brain **59**:135, 1936.

23. Hines, M.: The Motor Cortex, Bull. Johns Hopkins Hosp. **60**:313, 1937.

24. Frostig, J. P.: Clinical Observations on Insulin Treatment of Schizophrenia, Am. J. Psychiat. **96**:1167, 1940.

25. Munro, D.: (*a*) The Modern Treatment of Craniocerebral Injuries with Especial Reference to the Maximum Permissible Mortality and Morbidity, New England J. Med. **213**:19, 1935; (*b*) Cerebral Subdural Haematomata, ibid. **227**:3, 1942; (*c*) Craniocerebral Injuries: Their Diagnosis and Treatment, New York, Oxford University Press, 1938; (*d*) The Diagnosis and Therapy of So-Called Post Traumatic Neurosis Following Cranio-Cerebral Injuries, Surg., Gynec. & Obst. **68**:587, 1939.

Frazier, of Philadelphia, in 1928 taught that the solid type of hematoma was more often encountered at autopsy than at the operating table, and in only 45 of the 310 cases in Munro's series was the hematoma "solid," the remainder being made up of a maximum of cerebrospinal fluid and a minimum of blood, the "hydromas" of Guilherme da Costa and Adson.[26]

One can assume that decerebrate rigidity consequential on the compression induced by an ephemeral, surgically removable "hydroma" is not of the same nature as the terminally rigid decerebrate state wherein multiple petechial hemorrhages are found in the corpus callosum, the midbrain, the pons and the medulla. Of the latter "type" Rand[27] remarked that, although an occasional patient may recover without sequelae, when the patient survives he remains spastic and shows mental retardation.

REPORT OF A CASE

L. W., a woman aged 40, on Sept. 23, 1937, was thrown against the roof of the cabin of a truck, striking the top of her head, and in the rebound shattered the "nonbreakable" glass in the rear window with the back of her head. A few hours later she showed a drunken, reeling gait with weakness in both legs and complained of marked dizziness, intense headaches and a "sensation of firecrackers bursting in my head." She was seen by a physician on September 30; a lumbar puncture was not done. After a twenty-two day stay in a hospital and a sanatorium she was taken to her home, where the headaches, the dizziness and the reeling gait continued. On December 20, in an "amnesic" state, attired only in underwear, she stumbled a mile to the home of a friend, and there fell into a sleep lasting one hundred and nine hours, from which she finally awoke with dizziness, headache and numbness of the right side, with inability to use her right arm and with a dragging of both of her legs. Throughout January 1938 her symptoms continued unabated, with complete weakness of the right arm and the right leg and with increasing "weakness" of the left arm and the left leg. On January 31 she was first seen by me. She showed dilated pupils, fixed to light directly and consensually, ptosis of the right lid, anesthesia of both corneas, right-sided deafness with a marked deficit of hearing in the left ear, an ataxic cerebellar gait when supported, with a questionable cerebellar syndrome of hypotonia, dysmetria and asynergia, due in Grant's explanation to supratentorial compression.[28] There was anesthesia for pain, touch and thermal stimuli of the left side of the forehead, the left side of the face, the right side of the neck, the right arm and the right side of the body—the crossed anesthesia of a pontile lesion. Stretch reflexes and clonus were present bilaterally. A linear crack in the right parietal region 2½ inches (6 cm.) long is demonstrable in the original roentgenogram of the skull. Four days later, February 4, with the patient increasingly sleepy (not stuporous), with a clinical temperature of 96 and sometimes 94 F., flash-

26. Guilherme da Costa, D., and Adson, A. W.: Subdural Hydroma, Arch. Surg. **43**:559 (Oct.) 1941.

27. Rand, C. W.: The Neurosurgical Patient, Springfield, Ill., Charles C Thomas, Publisher, 1944, p. 176.

28. Grant, F. C.: Cerebellar Symptoms Produced by Supratentorial Tumors: A Further Report, Arch. Neurol. & Psychiat. **20**:292 (Aug.) 1928.

light photographs were made of her to demonstrate her quadriplegia. On the evening preceding surgical treatment, February 6, the pulse rate was 55, the respiratory rate 16, the pulse pressure 15 and the temperature 95.6 F. These findings of pulse, respiration and temperature were in agreement with those noted four months previously at the sanatorium; the contest between the cerebrospinal fluid pressure and the cerebral arterial pressure was, after this long period, continuing, without obvious signs of medullary collapse. February 7, after local anesthesia had been obtained with procaine hydrochloride, bilateral temporal burr holes were made, following the usual 3 inch (7.5 cm.) coronal incision. The left decompression opening showed gray-green dura mater under great tension, which when opened by a cruciate incision revealed a severely contused and congested brain, which did not pulsate and which was tamponaded against the skull wall. The brain began to herniate through the decompression opening, and when a Frazier brain needle was inserted into the lateral ventricle, generalized convulsions began. The incision was closed, therefore, in four layers with black silk sutures. Trephining must in such a case be, ipso facto, bilateral,[29] and when a subdural hematoma is a possibility, there is much more to be gained by this procedure (burr holes) than there is to lose.[30] On the right side, a silver dollar decompression opening having been made, the surface of the edematous dura mater was incised with dural hook and canaliculus knife. Immediately a trapped, localized xanthoproteic cerebrospinal fluid hematoma, neither grossly bloody nor semisolid nor solid, measuring about 100 cc., gushed from the incised dura and flowed steadily. It appeared to come from above, parietally and temporally. The patient gasped, breathing rapidly and deeply, and the right arm and the right leg could be voluntarily moved slightly for the first time in eighteen weeks! The dura mater was adherent to the skull, and the brain (which resembled a shriveled dehydrated cauliflower), instead of being jammed into the decompression opening as on the opposite side, was found at a depth of ½ to 1 inch (1 to 2.5 cm.) inside the skull! The pathologic finding was a torn and lacerated brain with contralaterally a huge "hydroma" (fluid subdural hematoma), and the decerebrate state was obviously due to extremely high localized intracranial pressure malignantly squeezing the midbrain. "It is a fairly common occurrence for a subdural haematoma to displace the midbrain so far to the opposite side by a temporal pressure cone that the contralateral cerebral peduncle is forced against the margin of the tentorium. An actual groove across the peduncle may result. By implication of the corticospinal tract above its decussation, a homolateral hemiparesis or hemiplegia occurs" (Kubik). The postoperative convalescence was uneventful except for a twenty-four hour period of excitement; the sensory symptoms disappeared, and the sensorium became clear, although the memory of the events of the preceding five months was extremely hazy. Postoperatively the manometric pressures varied from 300 to 140 mm. of water, measured with Fremont-Smith tubes. Forty days after operation all reflexes were hyperactive, but there was no motor weakness, nor were associated reactions present; only a slight facial weakness on smiling remained, which within a year had completely cleared. At the time of writing, in 1945, the patient is alive and well, with no neurologic residua. Neither headache nor psychoneurotic complainings as part of a "post-traumatic general syndrome" have appeared. What few

29. Coleman, C. C.: Chronic Subdural Haematoma: Diagnosis and Treatment, Am. J. Surg. **28**:34, 1935.

30. Case Records of the Massachusetts General Hospital, New England J. Med. **229**:347, 1943.

symptoms have developed from time to time have responded to the methods suggested by Denny-Brown,[31] Symonds[32] and Cairns.[33]

COMMENT

The patient illustrated the principle of the dissolution of the nervous system, seen as "release" phenomena, with the dethronement of the higher hierarchies of Hughlings Jackson,[8] and by a combination of ipsilateral cortical injury and contralateral compression of the corticospinal tract, the midbrain, the cerebellum and the brain stem, from a "hydroma," this patient climbed down her own phylogenetic tree, with ontogeny recapitulating phylogeny. During this declination the higher centers were depressed to a point where the decerebrate state existed with (1) double pyramidal system spasticity, (2) bilateral presence of the reflexes of Magnus and de Kleijn, (3) preoperative temperatures of 94 and 95.6 F. (interbrain phenomenon) and finally (4) disturbances of pulse and respiration due to medullary compression, and these are the criteria of decerebrate rigidity.[10b] Is human decerebrate rigidity then a reversible state? Here it was; clinically speaking, it depends on no exact Sherringtonian "mutilation," since by a fortuitous combination of injury of the ipsilateral corticospinal and extrapyramidal pathways and compression of the contralateral corticospinal tract, of the temporal lobe and of the brain stem, the criteria of the brain stem animal (cerebrum, thalamus and midbrain removed) were duplicated for eighteen weeks! Homologies between the condition of the cat mesencephalically transected between the red nucleus and the vestibular nuclei and the decerebrate rigidity of man are difficult to make on account of the wide gulf separating their phylogenetic strata; a chronically decerebrate cat of Bazett and Penfield[11] lived for two weeks, while herein described is a patient who "existed" for eighteen weeks, with recovery when the applied force was released. When it is further noted that this patient made a complete recovery, the sole recovery from the decerebrate state (so far as I have been able to determine) reported in the literature, and that six years later exhaustive clinical and electroencephalographic studies revealed only minimal neurologic residua, the impermanence of this particular dissolution of a human nervous system and of a state of human decerebrate rigidity is shown. The condition of decerebrate rigidity in man with tonic cervical reflexes is produced clinically, accord-

31. Denny-Brown, D.: (*a*) On the Nature of Postural Reflexes, Proc. Roy. Soc., London, s. B **104**:252, 1929; The Sequelae of War Head Injuries, New York, Philosophical Library, Inc., 1943, pp. 9-50.

32. Symonds, C. P.: Mental Disorder Following Head Injury, Proc. Roy. Soc. Med. **30**:1081, 1937.

33. Cairns, H.: Rehabilitation After Head Injuries, New York, Philosophical Library, Inc., 1943, p. 60.

Fig. 2.—Reflexes of stance; the tonic cervical and labyrinthine reflexes of Magnus: Supratentorial compression of the brain stem.

ing to Bailey,[34a] only by supratentorial tumors which compress the brain stem above the tentorium and thus release the bulbopontile mechanism on which this condition depends.

Figure 2.—By changing the position of the head, thus stimulating the cervical receptors, and by reorienting the head in space, modifying the labyrinthine reflexes, different attitudinal reflexes of Magnus are shown; these are tonic reflexes which mobilize the body musculature to follow the lead of the head. They last until the head assumes or is moved into a new position, and they are practically indefatigable. In figure 2 the head is turned to the primary hemiplegic side, with the body musculature then following the lead of the head with attitudinal reflexes; i. e., the left elbow and the left shoulder are fixed; the left arm, in excessive flexor tone, remains in its new position until the head is turned back to the midline. These are tonic cervical reflexes, which are indissolubly linked with labyrinthine reflexes. The left leg is extending, sliding over the floor, and the positive supporting reaction of Magnus is developing in the left foot. When the head is turned to the opposite side, i. e., to the side of the patient's secondary hemiplegia or left side, a similar condition results, opposite in character, of course; i. e., the right flexor-paralyzed arm assumes tremendous flexor tone. The maneuver shocks the patient greatly, leading to projectile vomiting with a "wide" pulse pressure. In figure 1*b* the head turns to the side with secondary hemiplegia, the left side, since the tonic cervical reflexes in this case of ingravescent quadriplegia are now present bilaterally. Magnus[1] and his co-workers[35] found that three factors are present in postural tone: (1) the local static reaction, in which only one part of the limb or the body is involved; (2) segmental static reactions, which involve a segment of the body, i. e., both hindlimbs, both forelimbs or the neck; (3) general static reactions, in which more than one segment or even the whole body comes into action. Both nociceptive stimuli and passive changes of the head modify extensor tonus of the limbs, and labyrinthine righting reflexes originate in the otolithic maculas of the labyrinth and act on the muscles of the neck. Symmetric righting reflexes arise from the maculas of the saccular structures in the labyrinth.[2] The left limb here has a dual function; not only is it a scratching and fighting weapon, but it also serves as an extended pillar for support, a function accomplished by a local static reaction. The stimuli necessary to evoke these reactions may be: (1) exteroceptive, the magnet reaction, caused by touching the foot, markedly exaggerated in decerebellation,[6] and (2) proprioceptive, evoked by the dorsiflexion of the distal parts of the limbs, which thereby set up a complex "stretch" reflex involving not only the "stretched" muscles, but the co-contractor, extensor, flexor and adductor muscles, around each joint for standing. Sherrington[5e] showed that in the positive supporting reaction the synergistic functions of reciprocally innervated antagonists fixed the joints, and not the stretching of the extensors. A point has been made by Rademaker[6] and others that in true decerebrate rigidity these head changes give rise to reaction chiefly in the extensor muscles, i. e., excessive "stretch" reflexes, but that in midbrain and in thalamic preparations both flexors and extensors are influenced simultaneously. Thus, positive and negative supporting reactions are found in midbrain and thalamus preparations, while in the experimental decerebrate animal these reactions are not

34. Bailey, P.: (*a*) Intracranial Tumors, Springfield, Ill., Charles C Thomas, Publisher, 1933; (*b*) p. 165.

35. de Kleijn, A.: Experimental Physiology of the Labyrinth, J. Laryng. & Otol. **38**:646, 1923.

present. With cerebral decortication, on the other hand, these supporting reactions, although not completely absent, are disturbed.

Figure 3.—The stretch reflex, the labyrinthine reflexes of Magnus and de Kleijn and the proprioceptive reflexes of the long muscles of the neck are the tripod in the production of decerebrate rigidity. The patient is in a state of decerebrate rigidity, lying supine, with the arms at the side and the lower limbs extended. The legs are then slowly elevated by the examiner and slightly flexed, whereat they remain in this position by virtue of plastic tonus for several minutes. The right leg, by a series of alternating movements, is lowered in a grotesque caricature of a normal movement; the left leg sinks in slow jerks. The passive flexion of both legs, be it noted, caused a violent flexor jerk of the left arm, a

Fig. 3.—Stretch reflexes; local static reactions; clonus; associated movements.

phenomenon which Brain [17] stated he could never observe. The right leg developed extreme rigidity and with a rapier-like movement, due to synchronous discharge of motor units,[36a] shook for many seconds so fast that a flashlight camera could not photograph it clearly. The optimum grade of hypertonus was obviously present,[36c] the hypertonus being brought to the optimum for clonus by postinhibitory

36. (*a*) Riddoch, G., and Buzzard, E. F.: Reflex Movements and Postural Reaction in Quadriplegia and Hemiplegia with Especial Reference to Those of the Upper Limb, Brain **44**:397, 1921. (*b*) Bard, P., and Rioch, D.: A Study of Four Cats Deprived of the Cortex and Additional Portions of the Forebrain, Bull. Johns Hopkins Hosp. **60**:73, 1937. (*c*) Viets, H.: Relation of Knee Jerk and Patellar Clonus to Muscle Tonus, Brain **43**:169, 1920.

exaltation of extensor rebound. When the alternating movements of the right leg subside, an attempt other than plantar stimulation is made to flex it, but it is impossible for a time by the exercise of any justifiable force to do so; the right foot is strongly flexed as a supporting pillar in a local static reaction of Magnus, "a positive supporting reaction," produced by concurrent and not by reciprocal innervation. By continuous proprioceptive stimuli to the foot, inducing a weak negative supporting reaction, the leg can be relaxed again. The stretch "receptor" is in the muscle, and its tone is a muscle back to muscle phenomenon, with one stretch afferent existing to four or five motorneurons.[21] In a series of studies of hemiplegia, in 1914 and 1919, Walshe [10a, b] drew attention to what he conceived as a similarity of the clinically observed "clasp knife" spasticity of hemiplegia to the decerebrate rigidity of Sherrington. Bard and Rioch [36b] regarded the rigidity as a cortical release phenomenon. Fulton [15] expressed the belief that the postural response giving rise to decerebrate rigidity is the "stretch reflex," in the elicitation of which one has tonus in the making. On the other hand, Pollock and Davis [7b] expressed the opinion that extensor rigidity is produced directly by labyrinthine reflexes without the existence of any tonus produced by the stretch reflex. As stated by Grinker [37] : "The differences in responses between reflexes in decerebrate and spinal animals suggests that the shorter latent period, reflex contraction and after discharge of the decerebrate reflex means that it is more under control . . . the function of higher centres is in some way inhibitory, to the final common pathway. . . . In decerebrate animals with vestibular nuclei intact, there exists extensor rigidity. A very rapid break occurs in this on nociceptive stimuli to produce flexion. The spinal animal, on the other hand, is flaccid and its response to nociceptive stimuli is a long continued depression of the stretch reflexes. Fulton conceives of the difference being due to the effect of the vestibular nucleus and its pathway on inhibitory influences to the final common pathway. It permits activity of the final common pathway with little interference from inhibitory influences. He conceives of the mechanism as an inhibition of the inhibitory pathway by a process of "long circuiting" up the cord of the afferent impulses to higher integrating centres, while in the spinal animal such long circuiting would be impossible."

Figure 4.—Associated reactions which are a composite of stimulus (voluntary tonic contractions), labyrinthine and proprioceptive cervical reflexes, are here completely stereotyped in form. There is no "obvious" paralysis of the left arm unless movements are demanded of the patient. Here she is asked to extend her finger toward a pencil, whereat the extensor antigravity muscles of the neck and the trunk come into play by segmental reactions. An associated movement of the right side of the face is beginning, the right leg characteristically extends, and the left equinovarus leg is seen extending off the floor as a prop in a supporting reaction. The finger never reaches the pencil, as flexor-extensor nullification occurs at the elbow, due to the body swaying forward to the plantigrade position—the quadrupedal standing of Brain.[17] The left arm goes into "clasp knife" rigidity, with the left elbow locked, and the trunk extensor antigravity muscles swing the body forward in an attempt to reach the pencil. Evidently, to perform this movement, antagonists must be relaxed, resulting in a latent period, the duration of which here is abnormally "long"; a stalemate between flexors and extensors is reached unless the body swings forward; on its doing so the arm extends, as Brain [17] showed in hemiplegic patients forced to assume the quadrupedal position, in the

37. Grinker,[2] p. 99.

Fig. 4.—Reflex erect standing; tonic cervical reflexes of Magnus; associated movements.

forelimb posture adopted by anthropoid apes. It was never necessary to have this patient in the plantigrade position to change upper extremity flexion to extension, as any tilting of the body forward set the arms extended as props by a local static reaction of Magnus. With the bending forward of the trunk to a quadrupedal position, the semiflexed arms (considered by Walshe [10] to represent a new prehensile function developing with the assumption of the erect attitude) showed, as Brain found, severe extensor tonus, locking the arm and converting it into a plantigrade prop representing, according to Brain, the evolutionary stage between man and anthropoid ape. Fulton [15f] asserted that the attitude of the upper extremities is in dispute because clinical observers have been unaware of the difference between high and low decerebration in primate forms.

Fig. 5.—Reflexes of crossed extension and Sherrington's reflexes of homolateral extension; associated reflex with no latency. Note the left arm in acute flexion, the head turning to the right. Compare figure 16, where the head swings left in advanced quadriplegia.

Figure 5.—Initially the patient's hands are resting on her abdomen. The examiner attempts to flex the left limb gently at the knee; the percussing hammer is seen poised over the patellar tendon. The popliteal space is evidently outside the "receptive field" (the flexor reflexogenous zone), and the leg extends refusing to flex, an example of a relatively rare occurrence, a homolateral extension reflex. In a spinal animal the extensor thrust is elicited by a stimulus to the sole of the foot and is a quick extension movement of the corresponding limb, usually

Fig. 6.—The reflexes of stance of Magnus; tonic cervical reflexes and tonic labyrinthine reflexes; paraplegia-in-flexion and paraplegia-in-extension. Spinal man is seen on the right; decerebrate man, on the left.

with flexion of the opposite limb. Here flexion of the homolateral upper extremity invariably occurred if the leg was stimulated outside of the flexor reflexogenous zone. By an associated movement the head is twisting to the side of the primary hemiplegia, spastic flexor tonus develops in the left upper extremity and the body will roll over completely to the right by a "body on the body" reflex of Magnus if the stimulus continues. It was this phenomenon of a homolateral extension reflex elicited by stimulating the upper part of the thigh, the groin and the perineum which led Marie and Foix,[38] in 1912, to deny a protective function to reflex flexion, because the movement of extension, so far from removing the leg from the danger zone, takes it closer to it. This homolateral extension reflex is a sexual reflex, obtained by mechanical stimulation of the skin of the upper two thirds of the thigh and the perineum in decerebrate preparations.[39b] Creed, Denny-Brown, Eccles, Liddell and Sherrington[39] showed that there were exceptions to the rule that in the decerebrate preparation stimulation of a hind-limb causes flexion. Light massage on the belly of an extensor muscle exhibiting decerebrate rigidity sets up reflex contraction within it, and kneading of the muscle can also sometimes cause relaxation of it. The extensor thrust, part of "gallop" in the spinal dog, is elicited by stimulation of the planta. Two other reflexes excited from the same area, namely, the positive supporting reaction of Magnus, Blake Pritchard, and Schoen, and the spring reaction of the thalamic animal, may be closely related to the same reflex mechanism. In this patient mechanical stimulation of the skin over the upper two thirds of the thigh and over the perineum caused homolateral extension of the left limb; flexion of the limb favored its appearance. Another (third) reflex, in addition to those of crossed extension and homolateral extension, is an inhibitory-excitatory reflex arising in the muscles.

Figure 6.—The right is the primary hemiplegic side, with the right leg in extension a few seconds previously. Initially the patient sat (for clearness of photograph) with feet and legs together and each hand resting in her lap (the right with some difficulty on account of spasticity). The neck was then slowly retracted dorsally to the 45 degree angle of Magnus, to evoke by this passively imposed position of the head a corresponding reflex modification of the reflex posture of the limbs. "The raising and retraction of the head and neck increases the extension of the forelimbs and lowers the hindquarters, as when a normal animal looks up at a high shelf" (Magnus). As the head was gently retracted, the shocked condition of the patient was observed, and the head was immediately ventroflexed. The phenomena observed here may be the result of dual maneuvers which combined to neutralize each other. The left arm displayed a violent flexor jerk and the left leg an equally violent extensor tonus, the head twisted to the primary hemiplegic side, with extensor tonus developing in the right flexor-paralyzed arm and not typical mammalian flexor tone (observe the fingers), the right primary hemiplegic leg in extensor paralysis flexed violently, showing tonic reflexes of the limb on the limb and the patient began to vomit; the blood pressure, taken immediately, was 90 systolic and 70 diastolic, while the pulse rate was 160. Here are observed spinal phasic flexor withdrawal on the right with reflex extension

38. Marie, P., and Foix, C.: Les réflexes d'automatisme medullaire, Rev. neurol. **23**:657, 1912; Les réflexes d'automatisme medullaire et réflexes dits de defense: Le phénomène des raccourcisseurs, Semaine méd. **22**:505, 1913.

39. Creed, R. S., and others: (*a*) Reflex Activity of the Spinal Cord, New York, Oxford University Press, 1932; (*b*) p. 73.

Fig. 7.—The plastic quality of decerebrate rigidity: stretch reflexes; reflexes of crossed extension; the flexion and extension reflexes of the upper limbs in incipient quadriplegia.

Fig. 8.—Decerebrate rigidity: flexion and extension reflexes in incipient quadriplegia; bilateral local static reactions of Magnus: the crossed extension reflexes of Philippson; facial participation in associated movements by irradiation.

Fig. 9.—Decerebrate rage: successive induction; segmental and general static reactions of Magnus; aggressive vocalization; thalamic syndrome (?). "Each individual in the course of its ontogenetic development climbs its own phylogenetic tree" (von Baer).

on the left. These two reflex patterns cooperate for standing and stepping.[16] De Kleijn [35] showed that dorsiflexion of the head in animals causes extension of the forelimbs and relaxation of the hindlimbs, ventroflexion of the head resulting in flexion of the forelimbs and extension of the hindlimbs. The right-sided phenomena may be the primary change due to dorsiflexion, and the left-sided the result of ventroflexion; in brief, on the right the animal looks up at the shelf, and on the left, under the shelf. When rigidity is extreme, an admixture of flexor and extensor activity may occur and unnatural functional activity result; the rigidity is artificial and, as Bazett and Penfield [11] showed, may swing over into predominance of flexor tonus and not extensor.

Figure 7.—The left leg is extending in a supporting reaction as a prop. The state of hypertonus is here so marked that both leg extensors, in which the stretch reflex is easily evoked, and arm flexors, in which the stretch reflex ordinarily is not easily evoked, show the stretch reflex with distribution and grading. The flexor-paralyzed right arm is being gently extended from its reflexly acutely flexed state. Tension receptors in the muscles send impulses to the reflex centers resulting in "stretch," with asynchronous inhibiting impulses to the centers from end organs sensitive to excessive stretch. The head and neck turn to the hemiplegic side by an associated movement and righting reflex; there is an associated contractural grimacing of the right side of the face, and in the left leg extensor tonus develops which becomes extreme. Antecedently, the left arm has swung up in violent flexor tonus; the sequence is: right arm, right face contraction, left arm flexion, right leg extension, left leg extension. The plastic quality of decerebrate rigidity dependent on "lengthening" and "shortening" reactions is displayed.

Figure 8.—The flexor muscles, belonging to the rapidly contracting group of skeletal muscles, rarely show any contraction in response to stretch; here, however, the phasic component of the reflex is manifested. The flexor-paralyzed right arm sinks by a series of cogwheel jerks with "shortening" and "lengthening" reactions. There is marked flexor tonus in the left arm with marked extensor tonus in the right and the left leg, with bilateral equinovarus. By reciprocal innervation and contralateral extension Philippson's reflex of crossed extension occurs. The local static reaction of Magnus is displayed in both dorsiflexed feet; the patient is anarthric and decerebrate and has passed from the stage of right primary hemiplegia to quadriplegia. Walshe [10] observed clinically that the occurrence of a crossed extensor reflex depends on the existence of a high degree of reflex activity in the extensors, such as is found in the affected limb in hemiplegia and in paraplegia-in-extension. The flexion reflex dominates because it is a defense reflex and hence is more important in the economy of the animal. The spinal pattern is flexor because of the arboreal ancestry of the human being, which utilized flexion more than extension.[2]

Figure 9.—The two separable reflex systems of motor innervation, the flexor "phasic" spinal reflex protective system escaping from noxious stimuli and the extensor tonic system of postural reflexes, are here displayed in the anarchic state of midbrain stem release due to supratentorial compression. The patient was seated with feet together on the floor, and the examiner stimulated, with a nociceptive finger flick, the left receptive field. In Sherrington's "spinal cat" nociceptive stimulation of a hindfoot leads to ipsilateral flexion with crossed extension. In the decerebrate animal such stimulation gives in addition extension of the ipsilateral foreleg and flexion of the contralateral foreleg. "Man is no cat, much less a rabbit." [2] These segmental reactions of Magnus record an arboreal beast epic, for with the right arm flexing and fingers braced in extension to throttle or

"straight arm" the attacker, and with the left arm, platysma and cervical muscles defending the vulnerable jugular veins ("the instinct for the jugular veins") by depressing the chin, "the animal is in its species position for defensive action by virtue of a general static reaction."[1] The left leg, by reciprocal relaxation of antagonistic extensor muscles and violent reflex flexion, initiates the mass response, the "electric chair" phenomenon. In the right primary paralyzed leg further extensor tone develops by a crossed extensor reflex which will support the increased weight imposed by the withdrawal of one limb escaping from the "noxious" finger flick stimulus; in the flexor-paralyzed right arm increased tone develops, with bracing of the elbow and extension of the fingers; in the left arm marked flexor tonus develops; the right side of the face grimaces; the trunk turns to the primary paralyzed side with a head-neck-axial righting maneuver of Magnus, and an atavistic snarling screech echoes through eons of time, or, in Sherringtonian phrase, vocalization is aggressive. The associated reactions are clearly seen, an essential for the production of which is that the hindpart of the bulb be in functional connection with the spinal cord, i. e., mesencephalic transection. The peculiar paresis of the right side of the face (mimetic facial paresis) of the thalamic syndrome is well displayed. Although Sherrington[5] observed that simultaneous reflex movements in the hindlimbs, as seen here, are opposite in "phase," i. e., the crossed extensor response, Magnus[1] shows that this is a purposive segmental static reaction for support. Reflex flexion removes the stimulated limb; the limb with the crossed extensor response therefore has to carry the weight of the free or hindpart of the body, made possible only by supporting reflexes of the right scapula, fixed to the thorax, and fixation of the shoulder joint; then by myotatic bicipital reflexes evoked by the stretching of the biceps, fixation of the elbow with the appearance of extension occurs. Additionally the crossed extensor reflex, causing increased tone in the extensors of the other leg, develops. The muscles of the back also take part in this general static reaction of the whole body, the thorax leaning forward, causing the right limb which has been lifted up, to be extended, thus preventing the body from falling. This "reflex figure" is a compensating balance restorer; for the postural reflex, chiefly extensor, concerns itself with supporting the body in the new position in which the flexors have placed it, giving way in turn to the prepotent flexors. Stimuli in the environment produce the most potent of the reflexes, the flexors seizing the final common pathway, as no two antagonistic reflexes can occupy the path at the same time. By "successive induction," opposite reflexes, which are in competition with each other, will occupy the common pathway alternately, and successive induction is shown here. The knee jerk as in the "spinal" dog was first elicited by gently rubbing the skin over the patellar tendon, resulting in a minimum extension; a nociceptive stimulus then resulted in flexion, and, following this, the gentlest touch on the skin of the tendon resulted in marked extension. This complete general static reaction is dissolved not only by the cessation of the stimulus but also by processes for the loosening of the segments involved, which are set in action by the muscle stimulus of the stretching of the distal joint extensors. This is the negative supporting reaction found infrequently, if at all, in decerebrate rigidity, in which the overacting stretch reflex is the commonest phenomenon. The spinal pattern of upper extremity flexion is seen here; modern man, slipping on a dance floor, fractures his radius; ancient man, protecting his head and neck from clubs, took the brunt of the blows on his ulna. Babinski[40] showed that in paraplegia-

40. Babinski, J.: Réflexes tendineux et réflexes osseux, Bull. méd., Paris **26**: 929, 953, 985 and 1053, 1912; cited by Walshe.[10]

in-extension reflex activity is present in both flexors and extensors but that in paraplegia-in-flexion (spinal lesion) there is "phasic" reflex activity in the flexors only. According to Magnus [1b] and Rademaker,[6] the presence of positive and negative supporting reactions in experimental animals rules out decerebrate rigidity, these reactions occurring in thalamic preparations, and in the midbrain and thalamic preparations the flexors and the extensors are influenced simultaneously, with both positive and negative supporting reactions being observed. Obviously, if Rademaker's criteria [6] are to be accepted, a study of this picture at this stage of compression places it in the midbrain or the thalamus class: The marked pseudoaffective reactions suggest a thalamic rather than a decerebrate "preparation"

Fig. 10.—The capitulation of the upper hierarchies of Hughlings Jackson: supporting reactions and body righting reflexes of Magnus; flexion and extension of upper limb reflexes of Riddoch and Buzzard; Kinnier Wilson's criteria for decerebrate rigidity (overpronated hand and equinovarus); lengthening reactions of stretch.

—the Dejerine-Roussy thalamic syndrome.[3c] In addition to paralysis of the arms and the legs there are here mimetic paralysis of the right side of the face and paralysis of the right half of the tongue, arguing an interruption of the pyramidal fibers to the facial and the hypoglossal nuclei. Bard [16] observed that a strong nociceptive stimulus applied to a decerebrate animal may elicit responses expressive of anger or rage, the animal struggling, clawing and biting, with widening of the palpebral fissure; but he found that these pseudoaffective reflexes never amount to an effective action of attack or escape. The integration of the full expression of

anger depends on parts of the brain which have been removed or put out of action, for the full complement of bodily changes constituting rage is due to a discharge of subcortical origin. Rage as a type of patterned motor activity is dependent on a central neural mechanism situated in the caudal part of the hypothalamus; it is inhibited by the cortex; pseudoaffective reflexes are more marked in the thalamic than in the decerebrate animal.

Figure 10.—Destructive lesions of higher centers yield negative conditions which permit positive signs and symptoms to emerge from the lower levels (Jackson [8]). The principle of dissolution of the nervous system with midbrain release is demonstrated here. The patient's primary flexor-paralyzed hand is felt, and the palm is scratched. There result a flexor "clasp knife" rigidity of the right arm with lengthening reactions and extension of the fingers, "the over pronation" of Wilson,[12] an associated flexor jerk of the left arm following the lead of the head, a tonic cervical reflex and an associated grimace of the right side of the face. The spasticity of the right platysma can be seen, with a twisting of the patient's antigravity muscles to the right side by the segmental reflexes of Magnus. She rotated the body toward the examiner with the right elbow used as a fulcrum; marked extensor tone developed in the right equinovarus leg with a body righting reflex. The sequence of these allied reflexes was probably induced by a lowering of the threshold of each succeeding reflex by the one preceding it, and the extension reflex seen here was induced by the preceding opposite reflex of flexion, the negative successive induction of Sherrington.[5] Bailey [34b] in discussing a patient with a meningioma described similar associated movements of the upper but not of the lower extremities. The postural activities of this "thalamic" or "midbrain" animal in Magnus' scheme—(1) the one limb static reaction, (2) the one segment static reaction, (3) the general static reaction—all can be seen here in their entirety. The dorsiflexion of the right foot to form a pillar is a local static reaction. The exteroceptive stimulus applied to the right palm produces the positive supporting reaction, which manifests itself as a general static reaction. Macnamara and Gunson,[41] in 1915, drew attention to the presence of pain in these crossed responses, and Bard [16] stated that mesencephalic transection (decerebration) in cats does not eliminate all responses expressive of anger and rage. Some pseudoaffective reflexes, erection of the hair and widening of the palpebral fissures, occur but do not lead to effective action of attack or escape.

Figure 11.—"The deficiencies of decerebrate standing result from the absence of certain postural reflexes. There is postural contraction of flexors and diminution of extensors." In the initial posture of the supported patient the hands were on the thighs. The primary hemiplegic leg in extension is gently elevated, and a proprioceptive stimulus originating probably in the reflexogenous soleus muscle causes development of tone in the antigravity muscles, the left arm begins to flex, with the body beginning rotation to the right with a head-neck-axial righting reflex and optical righting reflexes. The one limb static reaction of Magnus develops into a general static reaction. The supporting reactions are the exaggerations of those found normally; they are produced by proprioceptive stimuli, and, as Rademaker [6] showed, exaggerations of the supporting reactions are found in decerebellation. Here the head turns to the right, with the left arm following the lead of the head. Righting reflexes consisting of labyrinthine righting reflexes, body righting reflexes acting on the head, body righting reflexes acting on the body and optical righting

41. Macnamara, E. D., and Gunson, E. B.: Some Cases of a Crossed Reflex Associated with Pain, Brain **37**:408, 1915.

Fig. 11.—The righting reflexes of Magnus; head-neck-axial reflex; optical righting reflexes; tonic cervical reflexes.

Fig. 12.—Static reaction of Magnus; decerebrate rigidity and quadrupedal standing of Kinnier Wilson illustrating two criteria; overpronation of the hand and equinovarus of the feet; flexion and extension reflexes of the upper limb in quadriplegia.

reflexes (usually not found in a thalamic or a midbrain animal, since they depend for their presence on the integrity of the occipital lobes [15f]) are displayed.

Figure 12.—The examiner presses the palm of the patient's right hand, which is in flexor paralysis, in an attempt to produce the upper limb reflexes of flexion and extension of Riddoch and Buzzard.[36a] This sets in motion proprioceptive reflexes throughout the body (the camera shows the initial movements), and in easily observed sequence there occur static reactions of Magnus in the right wrist, the right elbow and the right shoulder; then follow locking of the jaws, fixation of the scapula to the thorax, rigidity of the neck and a leaning forward of the thorax, producing quadrupedal standing of Brain.[17] There are now fixation of the right knee with dorsiflexion of the right foot, giving it a prop appearance (equinovarus), fixation of the left scapula to the left thorax, fixation of the left elbow and of the left wrist with extension and overpronation of the fingers, extension of the left leg with introversion (a positive supporting reaction) and a nonphasic postural response which fixes the limb as a rigid pillar, the exciting stimulus being the stretch of the muscles which ventroflexes the joints. These robot-like reactions do not proceed with smoothness but appear awkward, with a long latent period, and are a grotesque caricature of a normal load-lifting strain. The upper limb reflexes originating in the hand activate the wrist extensors, which normally synergistically contract to prevent simultaneous flexion of the wrist; here they cause further extension, and the hand is bent backward, these movements resembling the reflex movements of the lower limbs in paraplegia. The extensor attitudes of the upper limbs, despite the typical right flexor paralysis and marked flexor tone, are of interest in the light of cases of hemiplegia observed by Brain [17] in which the patient was made to assume the quadrupedal attitude. Brain expressed the belief that decerebrate rigidity in the carnivores is reflex quadrupedal standing and that in man the hemiplegic posture represents reflex erect standing; the semiflexed posture of the arm in hemiplegia he held to be identical with the position of the forelimbs of some apes in a resting position. The truth of Brain's contention was obvious *bilaterally* in this patient. Blair and McDowall [42] in 1933 drew attention to Sherrington's observation that in monkeys during anesthesia flexor tone may develop in the upper extremities.

Figure 13.—The flashlight photograph shows the left patellar tendon being percussed. A marked extensor response occurs, due to inhibition of the inhibitory pathway by "long circuiting" up the cord of the afferent impulses to higher integrating centers, and the head is turned to the right; there is an associated movement of the right side of the face; the antigravity muscles of the trunk twist the body to the right, and in the left arm severe flexor tone develops, following, as Magnus [1] has pointed out, the lead of the head, tonic cervical and labyrinthine reflexes inducing a flexor jerk of the left arm, and if stimulation is continued, a body righting reflex will roll the body completely to the right. Exaltation of the knee jerk and crossed extensor reflex with diminution of the flexor reflex occur in the decerebrate cat or dog; the knee jerk is a spinal reflex

42. Blair, D. M., and McDowall, R. J.: On the Cortical Flexor Tone in the Forelimb of the Cat with Observations on the Hemiplegic Attitude in Man, Brain **56**:99, 1933.

Fig. 13.—Tonic cervical reflexes; the knee jerk and weakening of the extensor protecting suprasegmental augmentative influences.

varying with the degree of muscle tonus and in the decerebrate animal is the result of an asynchronous volley of impulses due to branching in pathways of the central axons. With the vestibular nuclei intact in decerebrate animals, there is extensor rigidity; on nociceptive stimuli flexion occurs.[2] (See figures 9 and 16.)

Figure 14.—A minimal nociceptive stimulus to the sole of the foot of the left leg produces body righting movements of Magnus exaggerated to gargantuan proportions and never under motor control. The flexion reflex of the left leg, beginning with flexion of the knee, with preliminary flexion of the great toe and extension of the foot, a support reaction studied by Balduzzi in infants up to 6 months and described by Schwab[43] as a negative supporting reaction, to be then

Fig. 14.—Tonic postural reflexes (associated movements) with head-neck-axial righting phenomenon of Magnus.

succeeded by the extension of Babinski, is to be seen. As Walshe[10] and Riddoch and Buzzard[36a] have pointed out, Babinski regarded the extension of the great toe in lateral plantar stimulation as a distinctly separate and cryptic reflex, an inexplicable sign of involvement of the pyramidal tract; the sign of Babinski is simply part of the reflex flexion of the whole limb, and it is devoid of any singular significance. In this patient extension of the great toe was commonly preceded by preliminary flexion bilaterally. There can be seen in addition

43. Schwab, O.: Ueber Stuetzreaktionen (Magnus) beim Menschen, Ztschr. f. d. ges. Neurol. u. Psychiat. **108**:585, 1927.

Fig. 15.—Righting reflexes of Magnus; reflexes of crossed extension; reciprocal and coincidental innervation; tonic reflexes of the neck and limbs on the limbs; Philippson's reflex.

Fig. 16.—Ultima Thule: bilateral tonic cervical reflexes, with head swinging to the left toward the side of the secondary hemiplegia; ingravescent quadriplegia. Compare previous photographs with head-neck-axial righting maneuver of Magnus directed to the right.

a twisting of the trunk to the right side with the associated grimace of the right side of the face, including closing of the eyelids, a body-righting reaction of Magnus. The postural reflexes are here overcome by phasic reflexes; as soon as flexion is over, the tonic reflex in possession of the limb restores it at once to its original posture, i. e., extension, by virtue of extensor "rebound." With extensor tone increased by reason of postinhibitory exaltation, on repetition of the minimal nociceptive stimulus there is shown marked reflex postural tone in the antigravity trunk muscles, and the patient then rolls completely to the right. This head-neck-axial righting reflex has been described by Schaltenbrand [4] as occurring in second year infants learning to walk.

Figure 15.—The initial position of the patient is supine, with the arms at the side; an intense degree of reflex excitability is present as the examiner stimulates the left tibial receptive field in an attempt to elicit a reflex from a tibial reflexogenic point of the lower part of the leg. The fingers of the right arm, the primary flexor-paralyzed arm, can be seen with extensor tonus (quadrupedal extensor reflex of Brain [17]) developing, and there is violent flexor contraction of the left arm, with the antigravity muscles of the jaw, the back and the abdomen rolling the body to the right side, the head and neck having previously led the way. The flexion reflex of the left leg is defensive and, being strongly affective, is prepotent in competition for the final common path; the strong extensor tone of the right leg (with a positive Babinski reaction) can easily be abolished by applying a stimulus to the right foot, reflex flexion (competitive reflex) breaking through and replacing the tonic postural reflex by occupying the common path to the exclusion of the extensor.[39] A crossed extensor response (Philippson's reflex) of the right leg with a right Babinski reaction is seen. Although Philippson's reflex was first described (1905) as an extension of one knee evoked by passive flexion of the other in the "chronic spinal dog," Sherrington [5d] showed that it could be obtained in the acute decerebrate preparation and that the source of the reflex was proprioceptive and not exteroceptive. The crossed effect is the reciprocal of the ipsilateral, involving an internuncial neuron, and is easily disturbed by competitive reflexes; it occurs by double reciprocal innervation, and the crossed extensor reflex and the stretch reflex both utilize motor neurons from a "common pool." The flexion of the ankle joint, a local static reaction of Magnus, with the leg fixed as a pillar by co-contraction, is an exaggerated supporting reflex. With this local static reaction due to stretch, the reciprocal flexors do not relax as required by the law of reciprocal innervation; instead they co-contract to serve synergically as fixators of the joint, coincidental innervation. Schaltenbrand [4a] showed that the infant learning to stand and walk in the second year does so by rotating the whole body around its axis, a maneuver comparable to the head-neck-axial righting of animals, and here this primitive torsion (replaced in adult life by direct elevation of the head and trunk from the floor) is shown. If the stimulus is great enough, the patient will roll completely over, with all four limbs in extension.

Figure 16.—The photograph shows the patient in hypersomnia, a state considered by Fulton and Bailey [15e] to be due to midbrain pressure on the hypothalamus. The patient is not stuporous; both pupils are dilated and respond poorly to light. The initial position of the patient is supine, with knees and feet together and with arms resting at the side. The head is elevated slightly to prevent

vomiting. A nociceptive minimal stimulus to the prime threshold of the left reflexogenous zone produced initially plantar flexion of the toes, then a Babinski extension with reflex flexion of the left leg and a turning of the head to the left side with marked flexor tone of the left arm, which is flung up toward the head, while the right arm flexes on to the abdomen. The head in this quadriplegic patient no longer turns to the right; the right arm flexes before the left arm by crossed extension, and the head thus follows the lead of the right arm. The motor center is plurimuscular, and the flexors of hip, knee and ankle in this reflex flexion respond simultaneously as Sherrington [5e] showed in 1910.

Decerebrate rigidity was now a fact with complete double corticospinal release; bilateral tonic reflexes were present, and in the face of this ingravescent quadriplegia the patient was relieved surgically of the "hydroma" which had forced her to descend her own phylogenetic

Fig. 17.—The patient in 1945, after recovery.

tree. At operation no extensive surgical procedures were necessary. Following an incision of the dura and rolling of the head, gravity took care of the "hydroma."

SUMMARY

A patient with decerebrate rigidity who recovered was closely watched and studied extensively. This condition involving double corticospinal release and bilateral expression of the reflexes of Magnus and de Kleijn is an extremely rare one. The paucity, indeed, of clinical cases of decerebration has led to confusion in regard to the boundaries of this entity, for if one omits Walshe's cases of alleged "hemidecerebrate" rigidity in association with hemiplegia and Wechsler's cases of tuberculous meningitis, rejected by some, the reporting of a series of cases of decerebrate rigidity, with or without recovery, is a task obviously impossible of fulfilment. In an exhaustive study of the literature I have found

no reports of recoveries—nothing but "the terminal quadripedal rigidities." [13a]

This woman suffered a cone compression of her temporal and parietal lobes, and by way of the incisura both the cerebellum and the brain stem were squeezed, with gradual release of the bulbopontile mechanism, the patient passing through the various phases of the thalamic, the midbrain and the brain stem animal. Cases of so-called human decerebrate rigidity should be reported in detail, with attention not only to such thalamic, midbrain and brain stem phenomena as are present but with special attention to the quality of the rigidity, to the presence or the absence of positive or negative supporting reflexes and the presence of tonic cervical, labyrinthine and body righting reflexes of Magnus and de Kleijn. Morphologic descriptions of attitudes are meaningless. The majority of the cases of premortem quadripedal rigidities (Walshe [10g, h]; Grinker [2b]; Davis [7c]) are obvious cases of decorticate and not of decerebrate rigidity.[15f]

To neuro-otologists this case of human decerebrate rigidity should be of interest, since for several days there were displayed Walshe's [10b] criteria: (1) the presence of double pyramidal system spasticities with complete double release, (2) the bilateral presence of the reflexes of Magnus and de Kleijn and (3) hypothermia without evidence of bradycardia and bradypnea until very late, when the patient passed from the midbrain to the brain stem stage. This clinical state of an admixture of thalamic, midbrain and brain stem syndromes was anamnestically present for four months, during which time the positive and negative supporting reflexes of the midbrain animal changed to the presence of a weak negative phase. Importantly, the patient lived to tell the tale. The associated reflexes were not the usual transient contralateral associated movements found at certain times in hemiplegic persons but were indefatigable, omnipresent and gargantuan when the patient passed from "primary" right hemiplegia to secondary quadriplegia. She was historically in the thalamic (predecerebrate) state for at least fifteen weeks prior to operation, a fact which compares interestingly with the history of the chronic decerebrated cats of Professors Bazett [12] and Penfield, which were kept alive for two weeks.

Concerning fluid subdural hematomas, the autopsy surgeon knows how grievously these "hydromas" are misdiagnosed. This fluid type of subdural collection, incarcerated in the subdural space, is free to move anywhere within that space, producing remote signs and symptoms.[25] Nor should one expect that the textbook distinction between midbrain and medullary compression has value in surgical time beyond the static conveniences of didactic terminology. Penfield and Cone [44] logically

44. Penfield, W., and Cone, W.: Elementary Principles of the Treatment of Head Injuries, Canad. M. A. J. **48**:99, 1943.

explained the production of decerebrate rigidity following craniocerebral trauma by the fact that increased intracranial pressure caused damage by compression of the brain, possibly by squeezing the temporal lobe into the incisura of the tentorium or by pushing the cerebellum into the foramen magnum, producing two different syndromes, midbrain compression and bulbar compression. Continued pressure causes dilatation of the pupils, paralysis of both extremities, decerebrate rigidity and death.

This report adds a case of "ephemeral" decerebrate rigidity in Man to those previously reported, i. e., the cases of "hemi-decerebrate" rigidity associated with hemiplegia,[45] craniopharyngioma,[10] midbrain softenings,[46] teratoma,[13a] tuberculous meningitis,[9a] endothelioma,[19] suprasellar cyst [7c] and hydrocephalus with basal inflammatory meningeal adhesions.[47] Since in the tripodal criteria of decerebrate rigidity the presence of the reflexes of Magnus and de Kleijn is a sine qua non, the interest of this morbid condition to neuro-otologists is obvious.

2122 North Main Street.

45. Walshe.[10] Riddoch and Buzzard.[36a]

46. Manson and Ferguson.[19] Grinker.[2]

47. Grinker,[2b] fig. 136.

PLANNING THE RHINOPLASTY

SAMUEL COHEN, M.D.
PHILADELPHIA

IT HAS been rather unfortunate that rhinoplasty was originated in obscurity and its progress hindered by the disapproval, even antipathy, of the great mass of the medical profession. Each rhinoplastic surgeon not only made his own errors but repeated the errors of others. That was the situation when I first started doing this work at the old Howard Hospital in 1926.

Conditions have changed since then. At that time the mere shortening and narrowing of the nose or the filling of a saddle nose was satisfactory both to the patient and to the operator. Since then the public has become plastic conscious—in fact, excessively so. Today the public expects results that as yet are too often impossible to attain.

A patient may have a tremendous nose. The skin is extraordinarily thick, with secretions oozing out of the pores on the slightest pressure. She requests a small, thin nose. We tell her the difficulties and state that a nose too small will produce incongruities in relation to the other features of her face, and so on.

The great majority of people who undergo corrections of nasal contour do so because they are dissatisfied with their appearance. These people have made a study of their faces. They know the bad features. They look for faults postoperatively and in finding them still present or, worse yet, finding new faults, are disappointed. It follows, therefore, that the surgeons should note these faults carefully before operating.

Nasal plastic surgery today is being practiced by physicians of varied background and training. Not only are general plastic surgeons and otolaryngologic surgeons doing rhinoplasty, but general surgeons are trying their hand at it. Rhinoplasty has now become a fairly common operation. Those performing it, to get good results, must be well trained in internal as well as external nasal anatomy and physiology. In other words, it is the otolaryngologist who is best qualified to specialize in this work.

The routine now followed by many of taking a course in plastic surgery, learning the essential steps of rhinoplasty and then operating may lead some into a great deal of trouble and annoyance. Any result

Read at the combined meeting of the Philadelphia Laryngological Society and the Section on Otology of the College of Physicians, Philadelphia, March 21, 1945.

Fig. 1.—Slight nasal dorsal curve.

Fig. 2.—Simple humped nose.

that is not as near as possible to perfection leads to the patient's dissatisfaction. With this view in mind and with full realization that knowledge of rhinoplastics is far from perfect, I shall try to stress

what to me is the most essential aspect of the work. That aspect is preoperative planning.

No two noses are exactly alike; therefore, no two operative plans can be exactly alike. In general surgery the objective is to remove the pathologic anomaly; appearance is a secondary factor. In rhinoplastics the objective is chiefly one of changing the size and the planes of essentially normal structures; here the cosmetic result is of paramount importance. Therefore, careful planning must precede such an operation.

In planning rhinoplasty, it is important to study photographs made with the subject's face in various positions and casts of the face. To

Fig. 3.—Humped long nose.

me, the pictures serve chiefly as preoperative records. The patients almost always forget what they looked like before the correction. It is the study of the anatomic deviation in each individual subject and the projected correction that is important in planning.

A careful study must be made of the intranasal anatomy. Special attention must be given to the nasal septum. A minor septal irregularity is best ignored. Three deformities of the septum have a bearing on the plan of operation: First, a marked septal cartilaginous bulge, which will prevent narrowing of the cartilaginous nasal structure. This

has to be submucously resected during the operation, and that portion which cannot be resected must be detached, pushed centrally and held there. Second, a marked deviation of the perpendicular plate and the vomer, which may prevent the nasal processes from getting together, thus preventing narrowing of the bony bridge. This can either be removed submucously during the operation or can be fractured and pushed centrally during the operation. Third, a low anterior twist of the septal cartilage. This the surgeon may cross hatch and push centrally, or he may remove sections or even remove the twist entirely, and on completion of the rhinoplasty a piece of cartilage can be inserted

Fig. 4.—Nose large in all directions.

to give a straight cartilaginous bridge. Sometimes more septal cartilage can be removed several weeks after the rhinoplasty. The important thing is studying the nose and planning the steps of the rhinoplasty before operating.

From the rhinoplastic point of view the nose can be arbitrarily divided into three parts: (*a*) the upper or bony part, made up of the nasal bones, the nasal processes and the perpendicular plates of the ethmoid sinus; (*b*) the middle cartilaginous section, made up of the upper lateral cartilages and part of the septal cartilage; (*c*) the lower cartilaginous part made up of the lower lateral cartilages and the inferior part of the septal cartilage. This is an arbitrary anatomic

review. For deformity in any of these sections there are several accepted plans of correction. Corrections of the lower cartilaginous division offer the greatest problem, are often difficult and present results that are far from perfect.

I will not delve into the operative steps usually necessary in most nasal plastic alterations, such as the intercartilaginous incision and elevation of skin, the septocolumellar separation, the removal of the offending part of the nasal dorsum and the fracturing of both nasal processes. These are the routine steps common to almost all nasal plastic operations. Each nose has one or more special abnormal or unsightly features and on the proper correction of these special abnormalities, in addition to the aforementioned steps, a presentable result should be obtained. In many phases of surgical treatment of nasal contour a standardized technic has as yet not been accepted. This is especially noted when one is attempting to correct the tip. Here, there is no fixed technic—only many methods, all far from perfect. I have found it best in many cases in which the tip is not too abnormal to let it alone. I will speak more of the tip later.

In planning the act of fracturing the nasal processes, several thoughts must be kept in mind. 1. Is the width in the region of the inner canthi so wide that use of a saw will leave a bulging ridge after the fracturing? Sometimes it is impossible to get out far enough with the right-angled Joseph saw; then a chisel may be used with good effect. The controversy as to which is better, the chisel or the saw, has not yet been decided. Each has some advantages. The chisel is easier to use and less fatiguing to the operator but more dangerous to the lacrimal duct. The question is to be kept in mind.

In nasal planning, consideration must be given to the length of the nose desired and the possibility of attaining that length, to the width of the nasal base and of the nasal dorsum and to the nasolabial and nasoglabellar angles that one thinks will be suited to the particular face. The shape and the size of the future nostrils must be given consideration. The shape of the new nasal tip must be visualized. By "the tip" I mean not only the whole tip structure but particularly the area where the nasal dorsum meets the anterior end of the columella. A whole paper could be well spent on this phase alone, but time will not permit.

In planning corrections of the tip the following thoughts may come up. Shall one incise just below the lower border of the lower lateral cartilage? This is one method of approach. Sometimes, it is better, however, to make the incision a little above the lower lateral margin. This can be done if there is not enough tissue between the lower lateral cartilage and the margin of the nostril and if by incising too near the

margin one risks the possibility that, with healing, scar contractions will cause pulling upward of the border. However, if the tip shows isolated bulges, then incision just below the margin of the lower lateral cartilage seems best. Sometimes the tip and the nostrils are satisfactory, but a widening is present above them. Here, it seems best to separate the lower lateral cartilages from above downward and remove only the required section of each. In a very large tip, I find it better to make my first intranasal incision, not at the intercartilaginous line, but above it.

To repeat, the tip is the most difficult area in rhinoplasty. There are many variations of tip requiring correction, and each calls for individual study of the tip. In some instances the abnormality seems best corrected by subcutaneous exposure and resection of a section of cartilage alone. In others, it seems best corrected by removal of an appropriate section of the lower lateral cartilage with the lining (as originally discussed by Joseph of Berlin). In some cases it may be well to plan to remove almost the entire lower lateral cartilage on each side (submucosally).

Another difficulty is the postoperative dropping of the nasal tip. No matter what method of suturing the septum to the columella is used, whether the entire septum and its covering are sutured to the columellar structures in toto or whether the mucosa of the septum is sewn to the skin of the columella on each side separately, postoperative drop occurs, although slightly less with the latter technic. What are the causes of this postoperative drop? Is it only the later contraction of the new septocolumellar scar? Is it a pushing downward of too large upper lateral cartilages? Is it the tip itself, the connection of which with the rest of the nose has been broken by the intercartilaginous incision and removal of a section of the upper lateral cartilage, so that its mere drag causes postoperative drop? All these may play a part in the postoperative drops, but these drops must be taken into account when planning a corrective procedure. I usually plan for a 10 degree drop in all my cases and usually the end result, after the drop, is pleasing. Sometimes, later corrections are needed for an excessive drop. These postoperative drops, I have found on questioning, occur at the hands of all doing rhinoplasty. A postoperative drop of the tip may lead to an alteration of the nasal appearance; for instance, there may be an unwanted lengthening of the nose, a new low hump may appear, or excessive alar creases may be noted.

Planning a nasal correction should be done carefully and the planning repeated several times by seeing the patient a number of times before operating. Points not noted at the first study may be found at the second or the third study.

Fig. 5.—Depressed tip producing dish face.

Fig. 6—Simple saddle nose.

The nasolabial angle must be carefully studied preoperatively. If it is too acute, a dishface appearance is probably present. For years I have got some improvement by placing a sliver of cartilage in front of the anterior nasal spine at the completion of the operation, but this

Fig. 7.—Saddle nose and dish face.

Fig. 8.—Twisted nose.

was not too satisfactory. Now, I find, it may be necessary to insert in addition a sliver via the mucosa of the upper lip.

The subject with a large broad nose, very thick nasal tip and very thick skin with large pores, which often ooze secretion, can be improved. but attainment of a so-called perfect result is as yet questionable. One

should be careful not to produce too high an upper lip. In women, this can later be corrected by "cupiding" the upper lip. The difference in level between the ala and the columella must be noted beforehand. Excessive removal of the lower border of the septal cartilage may lead to septal perforation, which, unless visible externally, may not be of any deleterious consequence. A marked retraction of the columella may also result, causing an unsightly deformity. Excessive removal of the septal cartilage, especially in the region of the anterior part of the nasal septum, may give a retracted columella.

When I state that all the outstanding disagreeable features of a nose must be taken account of in the planning before correction, it is

Fig. 9.—Twisted nose.

understood that in correcting these so-called bad features, new bad features may arise and, in turn, must be prepared for and corrected. It is, therefore, a good rule never to correct only the obvious deformity—say the hump alone, or the broad bridge alone, or the broad tip alone—or to shorten a nose alone. The nose must be considered in toto as a single organ in its relation to the face.

The saddle nose used to be a simple matter. A cartilage or other transplant was inserted, and the surgeon was satisfied. Today, it is best to plan carefully beforehand what the steps will be. Is a strut, in addition to a dorsal transplant, necessary? Shall one enter by way

of the columella, the intranasal route or the glabular route? Is the nose so wide that simply inserting a transplant will not be pleasing? Here the steps are: elevation of the skin, fracturing the nasal process, displacing a central section and then placing a transplant. The wide tip must be narrowed to conform to the new nose. When one goes to the operating table, a complete operative plan must have been laid out beforehand.

In some cases it may even be better to plan a nasal plastic operation in two or more stages.

More important than having fine surgical ability (technical skill) are the understanding of the abnormal anatomy present in the particular nose and the planning of the proper steps to obtain a pleasing result. The planning and the execution of the steps of the correction are the important element in this work.

In bringing out these few thoughts, I believe that those who have become interested in rhinoplastics will follow me easily, whether they agree or disagree with some of the statements. To those who have not given the subject some thought, the statements may sound vague, but I hope they will stimulate to further inquiry in regard to this interesting work.

Spruce Street Medical Building, Nineteenth and Spruce Streets.

A REVIEW OF ONE HUNDRED CASES OF ACUTE AERO-OTITIS

MAJOR JOSEPH LENTINE
MEDICAL CORPS, ARMY OF THE UNITED STATES

ACUTE aero-otitis media is a major cause of disability among pilots and crew members of the armed forces. The increase in the number of cases is self explanatory when one considers the stress and strain to which the auditory organ is subjected in flight. Man is primarily a terrestrial being, and the airplane has imposed demands on the ears for which they were not anatomically or physiologically designed. Rapid ascents and descents demand versatility of the auditory organ because of changes of pressure which confront every pilot. The changes are sudden and frequent, and the positive and negative pressures must be equalized by the eustachian tube. On ascent the adaptation of the eustachian tube is relatively easy, but on descent it is difficult both physiologically and anatomically, owing to the flutter-valve-like action.

The present day therapy of acute aero-otitis media is varied, as is evidenced by current literature. Many therapeutic measures are advocated, indicating that minor confusion exists. It is possible that some explanation may be found in the fact that clinical findings differ markedly from case to case, depending on the duration of the symptoms and on the conditions under which the attack was precipitated. It is important not to assume that each case of aero-otitis is identical with all others. The assumption is false, and one may be led to administer inadequate and improper treatment. Signs and symptoms offer many clues as to the severity of the condition, and inquiries concerning the events which preceded the attack are of great aid in determining the therapeutic procedure. Treatment and management are dictated by the pathologic conditions present in the middle ear and in the tympanic membrane.

This review of 100 cases has no intent of originality. It merely presents observations and the results of treatment among pilots and crew members over a two year period. Prior to collecting the series the treatment varied, but finally a standard therapy was adopted, and this led to fewer sequelae. An attempt was made to list each patient in one or another of four groups to simplify therapeutic procedure and classification of the physical findings at the time of the original examination. No attempt will be made to review the pathology and the physiology of

acute aero-otitis, since Armstrong [1] has written a thorough treatise in his excellent book. However, some of the important observations and the type of treatment will be detailed.

The four groups shown in the accompanying table are self explanatory. Group 2 is further divided into two subgroups: hospitalized and nonhospitalized patients.

It is to be observed that not a single patient required myringotomy. In fact paracentesis is rarely necessary in acute aero-otitis. Another striking observation is that the condition occurred unilaterally. In only 1 instance was there associated pain in the opposite ear. It was transitory, and the tympanic membrane appeared normal. No adequate explanation can be given for the unilateral incidence as both ears undergo identical physical stress under changes of air pressure. Questioning of

One Hundred Cases of Acute Aero-Otitis Media

Group	Condition of Tympanic Membrane	Patients	Elapse of Time Before Examination, Hr.	Disease	Infection Present	Hospitalization
I	Marked retraction, foreshortening of landmarks, peripheral hyperemia and injection along handle of malleus	59	¼-2	Unilateral	None	Not hospitalized
II	Injection of entire membrane, which showed dusky red color probably due to hemorrhage between layers; landmarks accentuated; fluid in middle ear	33	18-24	Unilateral	None	17 hospitalized, 16 not hospitalized
III	Membrane perforated, with seropurulent secretion in middle ear and external canal	7	24-36	Unilateral	Usually rhinopharyngitis	Hospitalized
IV	Membrane red with a linear perforation and blood in the external canal and middle ear	1	2	Unilateral	None	Hospitalized

flight surgeons confirmed this observation. Several stated that each had seen a few patients with bilateral aero-otitis, but generally involvement was one sided.

SYMPTOMS AND TREATMENT OF INDIVIDUAL GROUPS:

Group 1.—The predominant symptoms were (1) dull pain in the affected ear, (2) impaired hearing, (3) a sense of fulness, and (4) tinnitis occasionally. Audiograms showed conductive deafness in the frequencies between 64 and 1024 double vibrations per second.

Patients who sought treatment immediately after a flight rarely suffered from complications. The therapy is simple and easily administered. Tampons saturated with 0.25 per cent neo-synephrine hydrochloride are inserted into the nostrils. Each is allowed to remain in

1. Armstrong, H. G.: Principles and Practice of Aviation Medicine, ed. 2. Baltimore, William Wood & Company, 1943.

situ for five minutes to obtain maximum shrinkage of the nasal mucosa. Suction is then applied to remove all secretions from the nose and the nasopharynx. The orifice of the eustachian tube is then shrunk with cotton pledgets containing 0.5 per cent cocaine hydrochloride, attached to an angulated wire applicator. The middle ear is next inflated with air by means of a catheter, or inflation is achieved with a Politzer bag. It has been found that the most effective method consists in having the patient hold a small volume of water in the oral cavity and synchronizing compression of the bag with the act of swallowing. The method appears to be far superior to that of vocalizing "k" or "chocolate". Hearing returns to normal, and pain is relieved. Mild heat in the form of a hot water bag or hot towels may be advised. In most cases it is not necessary, since a cotton pledget inserted into the external auditory canal assures a feeling of protection and warmth. The following day hearing is examined, and if it is found normal, no further therapy is given. Some patients may require several treatments.

Group 2.—The symptoms of this group were essentially the same as those of group I except that they were more severe. They were as follows: (1) pain severe and accentuated by swallowing, (2) marked hearing impairment and (3) tinnitis, severe and persistent.

The pain was described as sharp, intensified by swallowing and yawning. The patient would point to the angle of the jaw and state that it was "deep inside".

Examination revealed edema and marked congestion of the torus tubarius. The tympanic membrane presented a dusky red color, and the landmarks were hazy or absent. The dusky red color conveyed the impression of blood extravasated between the drum layers. Catheterization or politzerization was not often successful. Airflow was minimal and high pitched, and frequently rales were heard.

Audiograms showed conductive deafness and involvement of the higher frequencies. An average loss of 30 to 40 decibels was present between the frequencies of 64 and 2048. Frequently a sharp dip was noted at 4096. It is to be noted that on descent symptoms of this group began between 18,000 and 22,000 feet.

Early treatment of this group is important, since there is secretion in the middle ear. Organization of any exudate should be prevented by prompt and adequate treatment of the eustachian tube to establish drainage. It is in this group that permanent impairment of hearing may ensue as a result of adhesions within the middle ear. Subsequent attacks lead to further loss of hearing and chronic aero-otitis.

Two factors are probably responsible for the complications which are often seen: one, delay in seeking treatment, the other, failure of decompression while the subject is still in flight.

Negative pressure in the middle ear is due to rapid descents from lower to higher pressure which cannot be equalized by the eustachian tube because of the flutter-valve-like action. Once landing is effected, it is most difficult to overcome; however, in slow descents interchange of gases is most adequate in equalizing pressure differentials. Decompression should begin while the subject is still in flight. Pilots should be instructed to ascend to a higher level than that at which symptoms of aero-otitis occurred. Ascent to rarefied air followed by gradual descent will allow equalization of pressure between the external and the middle ear by minimizing the flutter valve action of the eustachian tube. The pilot is instructed to swallow intermittently during the descent.

It is conceivable that time-consuming measures are impracticable in combat, but throughout all phases of training preventive measures can easily be enforced.

The group was divided into hospitalized and nonhospitalized patients. There were 17 of the former and 16 of the latter, all receiving identical treatment.

The following procedure was carried out. Since otalgia was severe in this group, sedatives were promptly given, followed by a hot water bag to the affected ear. The nose and the torus tubarius were shrunk with 0.5 per cent neo-synephrine hydrochloride and 1 per cent cocaine hydrochloride. Catheterization or politzerization was next attempted and was followed by pneumatic massage with a Seigel otoscope. Relief in some cases was transitory, but subsequent treatments produced marked improvement. The treatment was carried out daily, and hearing was tested for progress.

Hospitalized patients showed complete recovery in seven to ten days; nonhospitalized patients showed delayed recovery, which extended over a period of two to four weeks. Rest appears to be an important therapeutic agent; however, it is not to be abused. Patients may be allowed regulated activity in the ward, and rehabilitation classes may be attended by the majority of patients.

Group 3.—The signs and symptoms of this group were not unusual; they paralleled those found in patients with suppurative middle ear disease. Significantly, all patients had evidence of acute nasopharyngeal infection. Reference to the table will show that an interval of twenty-four to thirty-six hours elapsed before treatment was sought. This delay undoubtedly permitted infection to extend into the middle ear. Pyrexia of 99 to 100 F. was present in most cases.

Audiograms were not significant; they were identical with those of patients with purulent otitis media.

Treatment of the group consisted of the usual measures employed in cases of purulent otitis. Each patient was given 2 Gm. of sulfadiazine

for an initial dose, followed by 1 Gm. at four hour intervals. Adequate alkali therapy was maintained throughout the course of the treatment with sulfonamide compounds. Four times a day the affected ear was cleansed with dry wipes, and eight drops of the following solution was instilled: sulfathiazole 30 grains (1.94 Gm.) in water to make 1 ounce (29.5 cc.).

The patient was instructed to lie prone for five minutes with the affected ear uppermost. Recovery in all cases was uneventful.

Group 4.—This group consisted of a single patient, whose case of aero-otitis is included because such cases occur infrequently. Treatment was simple and was directed toward preventing complications. A pledget of sterile cotton was inserted into the external auditory canal. The tympanic membrane healed and hearing became normal.

PROPHYLAXIS

A great deal can be done to prevent acute aero-otitis media by simple instruction of airplane crews.

Most pilots with an acute infection of the upper respiratory tract are hesitant to report to a medical or a flight officer. Enthusiastic desire to fly is paramount, and a "minor cold" is disregarded. They should be emphatically warned of the probable complication of disease of the middle ear.

The proper procedure of securing decompression while they are in flights should be explained in simple language, stressing the physiologic equalization of pressure through the eustachian tube. It is much easier to decompress the middle ear while in flight than to allow negative pressure to increase, make a landing and then ascend to a higher altitude. If the latter is allowed to occur, the trauma and edema of the vital structures increase, and the negative pressure in the middle ear is almost impossible to overcome.

Attention should be given to correction of underlying pathologic conditions of the nose and paranasal sinuses. They include the presence of nasal polypi, deviation of the septum, hypertrophy of the turbinates and infection of the sinuses.

SUMMARY

One hundred patients with acute aero-otitis media were classified into four groups, each of which represented a different stage of the disease. This classification simplified therapeutic procedure and helped to clarify the confused state of the treatment of acute aero-otitis media.

Boston.

Case Reports

TREATMENT OF COMPLICATED ACUTE SINUSITIS WITH PENICILLIN

CAPTAIN RAYMOND W. MONTO
AND
CAPTAIN PHILIP L. LYLE
Medical Corps, Army of the United States

IT IS our purpose in this paper to present and discuss the treatment of a patient who had acute sinusitis which spread to the extraocular tissues and the blood stream, with possibly early involvement of the surrounding bone and irritation of the meninges.

Before the advent of penicillin therapy, acute sinusitis complicated by orbital cellulitis, bacteremia, thrombosis of the cavernous sinus or secondary meningitis was accompanied by a high mortality rate. The administration of sulfonamide compounds, with or without operative intervention, while of a certain value, frequently left much to be desired.

REPORT OF A CASE

A white man aged 23 entered the hospital with complaints of severe headaches and swelling of the left eye. The latter was of only four hours' duration. For one month he had suffered from attacks of frontal headaches, which he was told resulted from sinus disease. He was treated with a solution of a salt of ephedrine in the form of nose drops and took acetylsalicylic acid by mouth for this condition. The treatment for headaches was continued at intervals, but roentgenograms of the sinuses were not taken.

Two days prior to his admission to the hospital, while on maneuvers, he suffered from a recurrence of the frontal headaches. Due to their severity he consulted his squadron surgeon and was again given analgesic drugs, without relief.

On the morning of his admission he awoke with fever and found his left eye swollen shut and his headache considerably worse. Further inquiry failed to reveal additional factors of importance in either present or past illnesses.

Physical examination revealed an acutely ill patient who was irritable and at times irrational and who complained of an excruciating headache. The temperature was 100 F., the pulse rate 110 per minute and the respiratory rate 25 per minute. His height was 5 feet 11 inches (180 cm.) and his weight 160 pounds (72.5 Kg.). The blood pressure was 110 systolic and 70 diastolic. The abnormal findings were limited to the head (fig. 1).

There was exquisite tenderness over the left ethmoid sinus and both frontal sinuses, which were dull on transillumination. The left eye was swollen shut, the conjunctiva was chemotic, and complete examination was accomplished with difficulty. Some proptosis was evident, with complete immobilization of the eyeball and gross diminution of vision. The fundus was normal. Inspection of the nose showed fiery red mucous membranes and a mucopurulent discharge. A specimen of this material was obtained for culture and smear. A heavy postnasal drip was seen in the pharynx. Two plus nuchal rigidity (graded 1 to 4) was found, and the reflexes were hyperactive. The remainder of the examination by systems was done, with negative results.

Fig. 1.—Drawing showing edema and proptosis of the left eye of the patient with orbital cellulitis complicating acute pansinusitis.

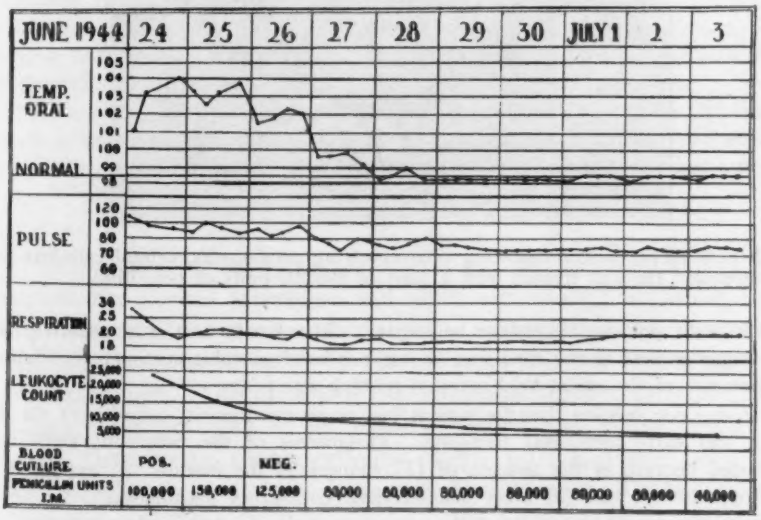

Fig. 2.—Graphic outline of the course of the patient with orbital cellulitis, acute sinusitis and bacteremia who was treated with penicillin.

The laboratory findings on admission were: hemoglobin, 90 per cent (Fisher photometer); red blood cell count, 4,590,000; white blood cell count, 22,350, of which 72 per cent were neutrophils, 15 per cent stab forms, 12 per cent lymphocytes and 1 per cent monocytes. The Kahn test for syphilis and the urinalysis gave negative results. Blood was taken for culture.

Because the signs and symptoms suggested meningeal and cerebral cortical irritation, a lumbar puncture was done at the time of admission. In an apparently relaxed patient the initial spinal fluid pressure was 300 mm. of water. The cerebral spinal fluid was clear, with a cell count of 12 white blood cells, all of

Fig. 3.—The patient following recovery from pansinusitis, orbital cellulitis and bacteremia. He was treated with a total of 895,000 units of penicillin.

which were polymorphonuclear leukocytes. The Pandy test was negative, and the sugar content of the fluid was 63 mg. per hundred cubic centimeters. Culture and smears of the spinal fluid revealed nothing significant.

It was our opinion that the patient had acute pansinusitis, cellulitis of the left orbit and early meningeal irritation. Thrombosis of the cavernous sinus was excluded because of the absence of (1) changes in the fundus, (2) extension to the opposite eye and (3) other characteristic circulatory phenomena.

The severity of the illness indicated the use of our strongest chemotherapeutic agent, penicillin. Surgical drainage was considered but postponed until the result

of the penicillin therapy could be determined. An initial dose of 40,000 units was given intramuscularly and 20,000 units every four hours thereafter. The remainder of the treatment consisted of adequate intake of fluids, sedation, nasal shrinkage and hot packs to the nasal sinus area.

Moderate improvement was noted at the end of twenty-four hours. The patient's symptoms were considerably relieved, but the temperature remained elevated. He was able to read Jaeger test type 10 with the left eye. A small localized mass was found in the region of the trochlea, and on aspiration 0.1 cc. of sanguinopurulent fluid was obtained. A small stab wound was made to facilitate further drainage, and the material was sent to the laboratory for culture and smear, the latter of which revealed many gram-positive cocci.

The condition of the patient was quite improved on the second day after admission. His temperature had fallen to 100 F. (fig. 2). Pus continued to drain from the nose; the drainage was more marked on the right than on the left, but that from the small stab wound was slight. The fundus was normal. The swelling about the eye remained unchanged. The laboratory reported a Staphylococcus aureus infection of the blood stream, the growth being slightly hemolytic, with approximately 8 colonies per cubic centimeter of blood.

Study of the pus from the stab wound above the eye revealed an organism identical in staining and cultural characteristics with that obtained from the blood. No predominant organism was isolated by culture of the intranasal pus, and a culture of the blood made on this date subsequently proved to be negative.

The patient continued to progress in a most satisfactory manner, becoming afebrile on the fifth day after admission. The dose of penicillin was then reduced to 20,000 units, given every six hours, and this dosage was continued for five days after the patient became afebrile (fig. 3). A total of 895,000 units of penicillin was administered to the patient during his hospitalization. On his discharge, twenty-nine days after admission, there were no residual signs of his disease and he was asymptomatic. Distant vision without correction was 20/15, and near vision was Jaeger test type 1 bilaterally. Six weeks after discharge he was completely recovered, and no symptoms had reappeared.

Roentgenograms of the paranasal sinuses taken on the third hospital day showed clouding of all the sinuses. A small area of questionable osteitis was located in the floor of the left frontal sinus at the junction with the ethmoid bone.

Progress films taken on the ninth hospital day showed clearing of all sinuses, especially of the right antrum. Roentgenograms taken two weeks later showed no abnormality except for some thickening of the membranes in the left antrum.

COMMENT

The diagnosis of simple acute sinusitis is not difficult, as in most instances the clinical symptoms and signs are classic, but when complications occur it is more confused. Acute meningitis is frequently preceded by symptoms referable to the nasopharynx but is excluded by lumbar puncture. Thrombosis of the cavernous sinus usually produces circulatory changes both in the external tissues and in the fundus of the eye. Erysipelas, simple cellulitis of the orbit and trichinosis are to be considered in acute diseases about the orbit. Owing to the characteristic findings in this case, no difficulty was encountered in arriving at the correct diagnosis.

Prior to chemotherapy many considered surgical drainage to be the treatment of choice in cases of complicated acute sinusitis. The use of sulfonamide compounds limited the spread of infection and was a factor of safety for the patient on whom a surgical procedure was carried out. Kirby and Hepp [1] reported cases in which after treatment with sulfonamide compounds and surgical drainage combined failed to affect osteomyelitis produced by acute sinus disease cure was obtained with penicillin therapy.

Penicillin has been used locally in the treatment of acute infection of the nasal sinuses and found to be superior to gramicidin.[2] This is due to the fact that it is soluble in water, that it is able to penetrate tissues and that it is nontoxic and nonirritating. Keefer and his associates reported one of the few cases found in the literature in which complicated paranasal sinusitis was treated by parenteral injection of penicillin. They listed 1 fatal case of pansinusitis caused by Staph. aureus infection complicated with bacteremia.[3] Of 9 cases with Staph. aureus bacteremia reported by Lyons,[4] 3 resulted fatally despite the use of penicillin. In Sloane's case of orbital cellulitis sulfadiazine was used before penicillin therapy was instituted.[5]

SUMMARY

In a patient with acute pansinusitis complicated by orbital cellulitis, Staph. aureus bacteremia, possible extension to the surrounding bone and early involvement of the meninges, penicillin therapy alone produced a remarkably quick and complete recovery.

1. Kirby, W. M. M., and Hepp, V. E.: Treatment of Osteomyelitis of the Facial Bones with Penicillin, J. A. M. A. **125**:1019 (Aug. 12) 1944.

2. Crowe, S. J.: The Local Use of Sulfadiazine Solution, Radon, Tyrothricin and Penicillin in Otolaryngology, Ann. Otol., Rhin. & Laryng. **53**:227 (June) 1944.

3. Keefer, C. S.; Blake, F. G.; Marshall, E. K., Jr.; Lockwood, J. S., and Wood, W. B., Jr.: Penicillin in the Treatment of Infections, J. A. M. A. **122**: 1217 (Aug. 28) 1943.

4. Lyons, C.: Penicillin Therapy of Surgical Infections in the U. S. Army, J. A. M. A. **123**:1011 (Dec. 18) 1943.

5. Sloane, H. O.: Orbital Cellulitis Treated Successfully with Penicillin, J. A. M. A. **126**:164 (Sept. 16) 1944.

Abstracts from Current Literature

Ear

PREVENTION OF DEAFNESS IN CHILDREN. RUSSELL M. DECKER, Ann. Otol., Rhin. & Laryng. **54**:358 (June) 1945.

The United States Children's Bureau states that a minimum of 2 per cent of all school children are hard of hearing. Prevention is important, and the primary causes of deafness in childhood are pathologic changes occurring in (1) the middle ear, (2) the eustachian tube and (3) about the tubal orifice. In the middle ear the drumhead may be thickened, perforated or distorted by adhesions. The ossicular chain may be involved by granulations, necrosis and disarticulation. Edema of the mucosa may be inflammatory or allergic. The eustachian tube shares the inflammatory processes of the nose and the throat. There is tubal involvement associated with allergic, as well as with inflammatory, conditions of the nose and the throat. Strictures may occasionally develop. About the tubal orifice the hypertrophic adenoid is the most important predisposing cause, though the size is not as important as the location, those close to the tubal orifice being more dangerous than those hanging free from the pharyngeal vault. Tonsils involve the hearing either by obstructing the tube or by constantly transmitting infection to it, so that the small tonsil may be as great a menace as the large, depending on the amount of infection present. Nasopharyngeal discharge of pus and new growths may involve the tube. Early diagnosis of the trouble is important.

M. V. MILLER, Philadelphia.

EXTERNAL OTITIS IN THE SOUTH PACIFIC. ROY F. NELSON, Ann. Otol., Rhin. & Laryng. **54**:367 (June) 1945.

Cases of external otitis usually comprised 50 per cent and sometimes as high as 70 per cent of the cases in the field of practice of the author in his fifteen months' experience in the hospitals of the South Pacific. His observations led to the conclusion that there are several types, at least three of which are definitely distinguishable, and that there are variations in the incidence and the type from island to island and from season to season.

The first type is the "fungous ear." It is insidious in onset; itching usually precedes other symptoms by days, weeks or even months. The first discharge is serous; pain may be severe, may increase rapidly, and seldom subsides until proper treatment is given. Getting water into the ear is often the start, and the author warns against the habit of turning the face upward while in the shower, thus allowing the canal to fill with water. There is exfoliation, and this may vary from mere scales to complete denudation of the epithelium and exposure of the bony canal. There are apt to be many relapses. Usually there is little swelling and not much tenderness.

The second type comprises about one third of the cases. There is a history of progressive and often rapidly increasing tenderness and swelling of the canal, frequently extending to the periauricular tissues. The condition may resemble acute mastoiditis. Slight fever and leukocytosis may be present, and the patient may be mildly toxic.

The third type is evidenced by a vague history of discharge, mild soreness and fulness of the ear. The canal is coated with or filled with a soupy, pale greenish discharge having the characteristic acid, sour odor of Pseudomonas aeruginosa. Mixed infections are quite common, with combinations of the different

types. Lack of specially trained technicians, mediums and reference works prevented bacterial studies from being carried to definite conclusions. Various fungi and bacteria were found. Treatment was based on the concept of types. Thorough, painstaking daily dry cleaning under definite vision was a necessary part. Wicks saturated with a solution of metacresyl acetate (cresatin) were placed in the fungus-infected ears daily for two hours, but this sometimes had to be cut to one hour because the agent burned some skins. In some patients who gave a history of chronic infections and who had a scaly skin after signs of active infection had subsided, filling the canal daily with 2 per cent salicylic acid in 95 per cent alcohol for from seven to ten days, followed by applications of a bland ointment, seemed to prevent recurrences.

The cellulitis type responded to similar daily packing with wicks thoroughly impregnated with 10 per cent sulfadiazine ointment, which were left in for twenty-four hours, dry heat, and sedatives for pain as required.

The third type responded quickly to packing for ten minutes daily with 5 per cent sodium thiosulfate, followed by 2 per cent acetic acid for five minutes. This treatment had to be continued for several days after subsidence of symptoms. Painting with tincture of merthiolate seemed to prevent reinfection but had no influence as a treatment. In the mixed forms many variations and combinations of these three basic treatments were used.

M. V. Miller, Philadelphia.

Massive Suboccipital Punctures in Degenerative Deafness With or Without Otosclerosis. Max Meyer, Pract. oto-rhino-laryng. **5**:1, 1943.

Meyer presents a survey of 80 cases in which he performed massive suboccipital puncture. With a total of 79 patients or a total of 153 impaired ears, improvement was shown in 51 patients or in 94 diseased ears. Of these 51 patients, 28 had a diagnosis of otosclerosis, while the remaining 23 had a diagnosis of labyrinthine atrophy. Tinnitus was present in 108 impaired ears, and it was definitely improved in 58 ears, or 36 patients; of these patients, 25 had otosclerosis, while 11 had labyrinthine atrophy. Meyer stresses the importance of careful and accurate testing and advocates making extensive auditory graphs. Accurate diagnosis is extremely important, and continuous observation must be made of treated patients, with special emphasis on relapses after an initial improvement of hearing. He holds that there is an increase of intracranial pressure in many of these patients, with frank atrophy occurring within the labyrinth, and that a suboccipital puncture will release the pressure and relieve the effects. It is impossible to heal primary diseases that lead to degenerative deafness by his method of massive suboccipital puncture. It is only the symptoms of impaired hearing and tinnitus that can be improved. The degree of improvement is variable, but often very satisfactory. The permanence of results is, also, rather indefinite. In 12 of his subjects the improvement was maintained for more than one year; in some, for more than two years; but relapses occur frequently. In view of the relative simplicity of the operation, even transitory improvement is extremely valuable, and should relapses occur the puncture can be repeated.

Persky, Philadelphia.

Pharynx

Penicillin Therapy in Hemolytic Streptococcus Pharyngitis and Tonsillitis. Norman Plummer, Dorothy Duerschner, Harold Warren, Francis Rogliano and Ruell Sloan, J. A. M. A. **127**:369 (Feb. 17) 1945.

Forty-five cases of pharyngitis and tonsillitis due to group A (Lancefield) hemolytic streptococci were studied by the authors.

Twenty-eight patients received penicillin in doses of 15,000 units every four hours for periods of twenty-four hours to six days. Symptomatic relief began in eight to twelve hours. Cultures of materials taken twenty-four hours after penicillin therapy was instituted were almost always negative for streptococci. After dis-

continuance of the drug therapy the frequency of reappearance of the organisms was inversely proportional to the length of time the drug had been given. Relapses occurred in 4 of 9 patients treated for less than four days. None of 10 patients treated for six days had relapses, but in 1 streptococcic sinusitis and pneumonia subsequently developed.

Of 11 control patients treated with sulfadiazine, out of 8 who had many streptococci on original culture only 1 had a culture negative for streptococci.

Of 6 control patients without specific therapy, the 5 who had many organisms on original culture continued to have cultures positive for streptococci throughout the period of acute illness.

BAIRD, Wauwatosa, Wis. [AM. J. DIS. CHILD.]

Nose

MUCOCELE OF THE NASAL ACCESSORY SINUSES. W. W. WILKERSON JR., Laryngoscope **55**:294 (June) 1945.

Wilkerson reviews the history of mucocele of the nasal accessory sinuses. The signs, the symptoms and the differential diagnosis are discussed. Treatment consists of complete exenteration rather than mere incision or partial removal. The leaving of part of the wall of the mucocele usually results in its recurring. The surgical anatomy and the surgical treatment are discussed. Many operative procedures have been proposed, but the external approach through the floor of the frontal sinus and the orbital wall as developed by Lynch is the operation of choice. This operation is described and a case reported.

HITSCHLER, Philadelphia.

SPONTANEOUS EXPULSION OF A FOREIGN BODY BY TRANSMIGRATION THROUGH THE NASAL WALL AFTER TWENTY-EIGHT YEARS. GEORGE KELEMEN, Laryngoscope **55**:375 (July) 1945.

Kelemen describes a case in which a wooden splinter was embedded in a nasal cavity for twenty-eight years and then was expelled spontaneously through the nasal wall. Other cases have been reported in which internal structures have been perforated by foreign material but none in which the nasal wall was penetrated. The histologic appearance is shown. Partial calcification was present.

HITSCHLER, Philadelphia.

BERNOULLI'S THEOREM AND UPPER RESPIRATORY DRAINAGE. Z. WILLIAM COLSON, Laryngoscope **55**:444 (Aug.) 1945.

Colson presents an interesting paper on the physics of blowing the nose in relation to drainage of the sinuses. If Colson's work is correct, he has added an important conception to present knowledge of intranasal function.

Colson considers the nasal passages as Venturi tubes and has applied Bernoulli's theorem (both explained in his article). The gist of this is that in a structurally normal nose (where there is a certain amount of constriction of the passages near the sinal ostiums), blowing produces a sucking action on some (at least) of these sinuses, aiding in their drainage. This effect, however, is defeated if the nose is overventilated, i. e., atrophic or altered by overuse of shrinking solutions. This is another reason why radical removal of nasal tissue should be avoided.

HITSCHLER, Philadelphia.

Miscellaneous

DISTURBANCE OF THE FUNCTION OF THE SALIVARY GLANDS. A. C. FURSTENBERG and ELIZABETH CROSBY, Ann. Otol., Rhin. & Laryng. **54**:243 (June) 1945.

The authors divide patients with salivary dysfunction into three groups: those with no demonstrable organic lesion of the salivary glands but with signs of

nervous instability which indicate a psychologic state of the nature of a depression; those which show an obvious organic lesion of the salivary glands, and those which have a dysfunction due to some organic lesion which irritates or interrupts the central pathways of the secretory nerves. The first group requires the aid of the psychiatrist, the second group falls rightfully under the care of the otolaryngologist, and the third is of particular interest to the neurologist.

Detailed descriptions and diagrams of neuron arcs and nerve pathways involved in salivary secretion are given, together with a review of much of the work which has been done experimentally.

In the first group of patients the diagnosis is not difficult, but great care must be exercised not to say or do anything which will crystallize the patient's symptoms and so make him refractory to subsequent neuropsychiatric care.

In the second group there are some whose condition results from lesions of the end organs of the secretory nerves. This fact seems to have been substantiated by the appearance of normal salivary flow after the administration of pilocarpine hydrochloride or nitrate. The effect of the drug is augmented by the simultaneous ingestion of ammonium chloride. Electrical energy is generated at the nerve synapse and a steady electric current is produced which discharges the nerve fiber at the axon hillock. The electric impulse conveyed along the nerve is created by a chemical agent, acetylcholine, in the region of the nerve synapse, and this substance acts best in an acid medium. They recommend that pilocarpine hydrochloride or nitrate be given by mouth in 10 mg. doses three times a day, that the intake of alkalis be restricted and that the patient be given 3 Gm. of ammonium chloride with meals, three days "on" and two days "off," the pilocarpine salt being given steadily. In some patients a return of normal salivation has occurred after the medication has been kept up three to six weeks.

Organic lesions of the central nervous system may produce dysfunction of the salivary gland, but they are associated conspicuously with paralysis of other cranial nerves.

M. V. MILLER, Philadelphia.

EIGHTEEN CASES OF APHASIA STUDIED FROM THE VIEWPOINT OF A SPEECH PATHOLOGIST. JEANNETTE O. ANDERSON, J. Speech Disorders **10**:9 (March) 1945.

Eighteen cases of aphasia are presented with the diagnosis, the type of linguistic handicap and a discussion of the type of speech correction if this was given. The ages of the patients ranged from 4 to 59 years. The patients varied so greatly that no common conclusions could be drawn. However, the author states that it seems probable that the observations made concerning the group may be expected to hold true for aphasic patients or groups of aphasic patients of which these may be representative. In the 5 cases of disorders interfering with the development of speech the author observed that the same therapeutic measures were effective in each patient but that these must be adapted to each if they are to be efficacious. Patients with such disorders need individual rather than group training, because of: (1) their peculiar rates of learning, (2) their varying mental levels, which are in constant progress, and (3) their dissimilar emotional patterns. In regard to 13 adults with linguistic regression a number of inferences were drawn, some of which are as follows:

"1. Retraining of speech may be started successfully after the acute stages of the illness have passed.

"2. Retraining in speech seems to be useful in securing increased emotional stability for aphasic patients.

"3. An individual who has been familiar with linguistic symbols seems to be stimulated to produce oral language more rapidly and more accurately by written or printed symbols than by objects or pictures.

"4. A knowledge of ontogenetic development of speech is useful in retraining aphasic patients. . . . Nonsense syllables and phonics seem valuable only as they are fitted into this pattern.

"5. Intensive retraining in speech for a short period of time is beneficial for aphasic individuals; further training seems to lead to further improvement in speech."

The term "receptive" used in combination with aphasia needs explanation and specification before its application is either clear or useful in retraining procedures.

Further observations are:

"Speech retraining may be started successfully only after there is some spontaneous attempt at communication through overt expression, oral or otherwise. . . .

"In retraining procedures, the use of numerical symbols in conjunction with actual objects seems to facilitate counting, addition and subtraction.

". . . relearning of speech tended to follow the pattern of speech development ordinarily followed by children in learning speech. . . .

"Retraining in speech seemed to aid the patient in achieving greater emotional stability. . . .

"Printed and written symbols elicited more accurate and rapid oral responses . . . than did corresponding objects and pictures. . . .

". . . former levels of excellence apparently cannot be reached, although the activities to which the patient has been accustomed before the aphasia can be re-educated to some degree.

"Results of retraining in speech, up to this attainable level or plateau, seem in proportion to the patient's active cooperation and interest and to original training. . . .

"With even a supposedly circumscribed lesion of traumatic origin, there may be diffuse impairment of the cerebral mechanism for language. . . ."

Other more specific inferences are drawn in discussion of the individual cases.

PALMER, Wichita, Kan.

THE LOCI OF STUTTERINGS IN THE SPEECH SEQUENCE. SPENCER F. BROWN, J. Speech Disorders **10**:181, 1945.

Stutterers were asked to read twice a 1,000 word selection. All of the stutterings of all of the subjects were studied individually and after being classified as mild, moderate and severe, in relation to the phonetic value of grammatic function, position in the sentence and length of the words read. Subjects whose stutterings were classed as mild had almost no stuttering on words which did not have at least one of the four factors just mentioned present. The following conclusions were drawn from the data: 1. The presence of one or more factors was accompanied by a greater relative increase in percentage of stuttering. 2. The presence of each additional factor beyond the first one was accompanied by a fairly constant increase in the relative degree of difficulty. 3. There was a high rank-order correlation between amount of stuttering and degree of presence of the four factors. 4. Only 5.3 per cent of the stutterings recorded could not be accounted for in terms of at least one of the four factors. 5. It is chiefly in terms of these four factors that evaluations are made as to the prominence of each word in the speech sequence. This appears to be true of both stutterers and nonstutterers. 6. It is probable in terms of the foregoing evaluation that the loci of stutterings are to be accounted for. In general, stutterers tend to avoid words which are evaluated as being conspicuous and react therefore with caution, hesitancy, effort, conflict, etc. These reactions are predominantly stuttering as contrasted with normal or nonstuttering speech.

PALMER, Wichita, Kan.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

JOHN F. DELPH, M.D., *President*

WALTER H. THEOBALD, M.D., *Secretary*

Regular Monthly Meeting, Nov. 5, 1945

Significance of Positional Nystagmus in Otoneurologic Diagnosis. Presented by DR. JOHN R. LINDSAY.

Clinical observations of postural, or positional, vertigo and nystagmus were made in a series of approximately 100 cases of "pseudo Ménière's syndrome." In this syndrome the absence of auditory disturbances and of other neurologic findings has made the localization of the source of the vertigo indefinite. Both the examination for spontaneous nystagmus, made with the patient in the customary upright position, and the routine tests for vestibular function have usually failed to indicate definitely the location of the disturbance. On the basis of the reports of former observers, particularly those of Nylen on encephalic tumors and Seiferth on other otoneurologic conditions, it has been possible to differentiate two simple types of postural, or positional, nystagmus:

Type 1. The direction of the nystagmus changes with changes in position or changes while the head remains in the same position.

Type 2. The direction of the nystagmus remains constant but the nystagmus may appear only in certain positions or is influenced in intensity by changes in position.

Type 1 has been found only with lesions involving the central vestibular pathways, whereas type 2 has been found both with peripheral and with central lesions.

In the so-called pseudo Ménière's syndrome a postural type of vertigo is a characteristic finding. The postural nature is sometimes obscured during the severe stage of the attack but becomes apparent as the vertigo begins to recede, or it may be characteristic from the beginning of the attack. The observations in this series indicate that the positional nystagmus in pseudo Ménière's syndrome is of type 1 in the great majority of cases. Type 2 has been found in a smaller number. On the basis of information gained both from patients with demonstrable lesions of the peripheral vestibular apparatus and from patients with lesions of the central apparatus it is concluded that type 1 positional nystagmus is an indication of a lesion affecting the central vestibular apparatus; this type of nystagmus in pseudo Ménière's syndrome is probably good evidence of a central origin. Type 2 positional nystagmus, which is seen in some cases of pseudo Ménière's syndrome, might arise from either a peripheral or a central location in such cases.

Approximately one half of the patients with pseudo Ménière's syndrome were patients with an unusually low blood pressure and other symptoms of vasomotor instability, sometimes called the hypotensive group. In the other half of the patients the vertigo could be attributed to such causes as toxic disturbance or hypertension, and in many the cause was indefinite.

DISCUSSION

DR. SHERMAN L. SHAPIRO: As Dr. Lindsay's paper is a clinical one, perhaps the most useful comment I can make is to summarize my own experience. This comprises probably more than 1,000 patients in whom I have observed postural nystagmus. Most of them had head injuries, but there have also been a good

many who had pseudo Ménière's syndrome. I have used the Frenzel glasses for more than twelve years and consider them indispensable, because without their use I am certain a number of cases of postural nystagmus of mild degree would have been missed. In my experience this has been the most frequent objective finding in patients in whom vestibular abnormalities were present. The most common form has been the horizontal, and the most frequent direction was to the left.

Excluding medicolegal cases, my experience has been that when the patient complained of vertigo at the time of examination, a study with Frenzel glasses invariably showed some degree of postural nystagmus as well. I believe both phenomena are of vestibular origin and due in the great majority of cases to involvement of the central nervous system. I do not see how a peripheral disturbance can be diagnosed in the absence of marked loss of hearing or of otitis media. There are in my experience two types of postural nystagmic reaction; in one the nystagmus does not occur immediately but after a lapse of a few seconds, and is pronounced. In the second it comes on at once and is weaker as to both amplitude and duration. It would seem that these two varieties are somewhat different in mechanism, but to say that the first type, or true postural nystagmus, is due to otolithic involvement is to take a good deal for granted, particularly as no one has produced nystagmus by otolithic stimulation. I have not been able to classify my cases as to types, possibly because my material included relatively few tumors. Most of the patients I have seen showed type 2 nystagmus; yet in the great majority of them this has been unmistakably of central origin. Toxic and other irritative factors, mentioned by Dr. Lindsay, are of course important, but more important are circulatory changes, that is, arteriosclerosis, hypertension and, in head injuries, vasomotor changes. In nearly all cases of the last-named type I have found postural nystagmus at the outset. However, in most instances, if the patient is followed, the nystagmus is seen to disappear later, as the vasomotor centers take control. There is no doubt in my mind that the underlying disturbance is a transient type of anemia or of hyperemia which momentarily affects the vestibular center.

I think we have been privileged to hear a timely and able discussion of this important subject, and one which will be of practical benefit to every otologist.

Dr. E. J. Blonder: Dr. Lindsay has been courageous in taking up this subject. A few years ago I went over the literature rather thoroughly; it contains many controversial reports. Otologists from Bárány on often speak of positional nystagmus as having its origin in the semicircular canals. Physiologists, as well as those doing experimental work, as a general group consider positional labyrinthine reflexes due to otolithic stimulation. However, otolithic reflexes have never been demonstrated experimentally to produce nystagmus.

The reason there is much clinical discrepancy in the literature is that there is relatively little experimental work on lesions of portions of the labyrinth, with these lesions being correlated to physical findings. There are great technical difficulties in producing these isolated lesions in a live animal. Versteegh reported section of the utricular nerve of only 1 rabbit. Magnus and DeKleyn did some fine original experiments on positional labyrinthine reflexes.

As to Dr. Lindsay's method of noting positional nystagmus, the physiologists would not be in accord with him in regard to his method of recording nystagmus immediately after the change in position, but would prefer to note the reading after a short interval of time, to make sure that the reflex observed was due to position, not to the motion caused by changing position. Examining a large number of patients with verified intracranial lesions, I did not record anywhere near 90 per cent with positional nystagmus; on the contrary, only a few responded with positional nystagmus. I had the opportunity to review labyrinthine tests in histories in a number of institutions, and I attempted to correlate the results of the tests with operative intracranial findings. One finding I have in mind was a tumor of the pons; five or six years previously an otologist examined the patient and

found no nystagmus on ordinary caloric stimulation but was able to bring out nystagmus by irrigating the ear with hot and then with cold water. There is a question whether that type of observation is of much value in diagnosis. To me positional nystagmus alone has not much significance in localization unless an otoneurologist or a neurologist expert in the pathology of the brain can bring out additional findings which may be correlated in arriving at a diagnosis. Otherwise I should just note that in certain positions the patient has nystagmus. If a patient was toxic or had vasomotor disturbances, how could one be sure whether the effect was referable to a lesion in a central or in a peripheral location?

DR. ALFRED LEWY: The members are indebted to Dr. Lindsay and Dr. Shapiro for clarification of a confused subject. I agree with Dr. Blonder on that. I should like to ask Dr. Lindsay whether he has correlated the caloric reaction with the postural nystagmus. Is there any constant relationship? In the case of hypotension was the blood pressure taken before and after the test? I have seen 2 cases of organic heart disease in which I believe hypotension caused the vertigo. That could not be proved, of course, except therapeutically; therapeutic measures to improve the heart action improved the vertigo.

I should like to have this dissertation presented to some industrial surgeons. Some are inclined to believe that all disturbances are due to pure psychoneurosis or malingering or a desire for compensation. That question is sometimes difficult to answer, and I wonder if Dr. Lindsay ever found objective evidence, such as postural nystagmus, in cases of pure psychoneurosis. I disagree with Dr. Blonder in the idea that unless the neurologist can spot the otoneurologic symptoms there is nothing to it. I know of a number of cases in which otoneurologic symptoms were the only objective ones present though the brain showed organic changes.

My idea that industrial surgeons should avail themselves more frequently of the services of the otologist before sending back to work a man with a head injury is based on a number of experiences. One of the large railroad companies elected to take the word of their chief surgeon in such a case, and the man was put back to work, perhaps with the idea that he was suffering from psychoneurosis. He quite promptly fell off a freight car and sustained another head injury with unconsciousness, and the railroad paid a substantial sum in settlement of the claim. They had had the advice of an otologist, who did not agree with the chief surgeon, but they elected to take the latter's opinion.

DR. HOWARD C. BALLENGER: I should like to stress one point in the etiology of pseudo Ménière's disease. In my experience in ordinary office practice in which acute infections of the upper respiratory tract and their complications form a large part, acute infections or congestion of the middle ear and the eustachian tube seem to be the most frequent cause. In a large clinic other etiologic factors, such as circulatory disturbances, would probably be found most frequently.

DR. JOHN R. LINDSAY: I wish to repeat that the primary object of this study was to try to determine if possible whether postural or positional nystagmus contributes reliable information which will help to differentiate a peripheral from a central origin of vertigo. During these observations type 1 positional nystagmus has not been observed in association with any known peripheral lesion, that is, one in which vertigo was associated with tinnitus or deafness. All positional nystagmus of known peripheral origin was type 2. In the patients with pseudo Ménière's syndrome postural vertigo and positional nystagmus proved to be almost constant characteristics, although sometimes obscured in the more severe stages. Type 1 occurred in the majority, but type 2 occurred in a few.

Evidence obtained from known lesions of the brain indicates that type 1 positional nystagmus occurs mainly in the presence of pontile and cerebellar lesions. Some tumors above the tentorium will also produce postural nystagmus. Nylen has noted vertigal nystagmus in the head-hanging position in such cases.

Many of the patients examined had spontaneous nystagmus in the upright position during some stage of the attack. The postural character may become evident in receding stages or may be characteristic throughout.

Regarding the question whether a certain toxic disturbance without other neurologic findings can be said to be central rather than peripheral in origin, it can be said that on the basis of the factual information accumulated so far it looks as though type 1 positional nystagmus is a reliable indication of a central origin and the majority of the group with pseudo Ménière's syndrome have had this type.

Frenzel's glasses are designed to eliminate the possibility of the patient fixing his gaze on objects in the environment and thereby suppressing nystagmus. They also give a better view of the eyeball.

No definite relation has been observed between the caloric responses and postural nystagmus. In the case of pseudo Ménière's syndrome the caloric reaction was routinely within normal limits. With tumors of the posterior fossa there are all sorts of variations in the response. For instance, the caloric response may not be present on one side before operation and present again after operation, whereas the positional nystagmus may be present before and absent after operation in the same case. There is no definite correlation that my associates and I have been able to observe.

The relationship of blood pressure to vertigo is an interesting question. It is known that there is a vasomotor response to stimulation of the labyrinth, and the question might be raised whether the low blood pressure was not due to the vertigo. But it seemed, so far as we could tell, that the low blood pressure was fairly constant throughout the period that the vertigo could be elicited.

I do not think that routine neurologic and otologic examinations have taken sufficient cognizance of postural nystagmus. It is one of the most useful tests for a vestibular disturbance. I have never observed positional nystagmus due to psychoneurosis. The presence of such nystagmus with the accompanying vertigo is probably a reliable indication of a true vestibular disturbance.

My associates and I have seen some cases, not many, of vertigo due to nonsuppurative disease of the middle ear, but these are classified under "Ménière's syndrome" because there is regularly some interference with auditory function. In our experience this has not been a large group.

Relation of Ocular Movements to the Vestibular System in Man. Presented by DR. HENRY PERLMAN.

The otologist is largely dependent on the studies of spontaneous and induced ocular movements in evaluating vestibular function. Because of this, a number of points are worth emphasizing.

1. The teaching of vestibular movements of the eye should include a consideration of all mechanisms involved in such movements and the common lesions of these systems that distort normal movement.

2. Because of the higher cortical organization of the visual system in man and the introduction of such factors as consciousness, volition, attention and discrimination, the clinician should study this problem in man. If there have been careful observations and recordings and these correlated with neurologic findings and postmortem or operative findings, conclusions arrived at can be valuable. In the words of the late Gordon Holmes, the method used should be the same as that of an experimental physiologist, which is to study the simpler components of a complicated function, isolating each of them and then attempting to integrate them into a complete picture.

3. The moving picture record of pathologic movements of the eye is valuable when properly interpreted. It can bring to the student in a short time the results of an extensive clinical experience. It can also demonstrate all normal movements for comparison.

4. The method used in this study for recording movements of the eye in man has certain advantages for the otologist wishing to investigate some of the problems in this field.

(*a*) It does not encumber the eye. (*b*) It can record movement of a closed eye. (*c*) It can be used with a patient on a stretcher.

The organization of the subject of the basic ocular movements in man is attempted in a moving picture, so that one may use this as a framework of reference for interpreting abnormal movements. This organization is assisted by the use of a number of general theoretic concepts. Nevertheless, lesions of the brain stem and the midbrain particularly will continue to tax all one's understanding. The result of a central lesion is not simple loss of function but the response of the remaining neural apparatus to the abnormal situation produced by the lesion, including disordered activity of the undamaged structures. Instead of the expression "nystagmus in direction of gaze" my associates and I use "inability to maintain lateral deviation." We assume that when the eye moves slowly toward center the movement is due to tonic muscle pull tending to keep the eye at center and to return the eye to center when it is moved away from center. When the eye quickly returns to the deviated position the return is considered the result of cortical stimuli that are adequate to move the eye but inadequate to maintain the eye in deviation. Again, as the cortex releases the eye and allows it to start sliding back slowly toward center, the cortical stimuli again become adequate and produce another quick movement. When the cortex releases the eye—anesthesia, coma, sleep—the eye assumes a more or less central position. When the eye is moved away from center mechanically, it slides back to center when released, indicating an underlying tendency for the eye to return to center—slowly—when released by the cortex.

One may consider further that the central position of the eye is due to symmetric tonus of the ocular muscles and that this symmetry may be disturbed by lesions of the brain stem and the cortex, resulting in an eccentric zero position for reference of movement.

Further, in the presence of spontaneous vestibular nystagmus, more marked movements of the eye are observed when the subject looks toward the quick component than when he looks toward the slow component. This may be explained by considering a super position of the slow vestibular pull on the slow tonic pull toward center. The addition of these two similarly acting stimuli results in a stronger movement for the cortex to overcome in maintaining deviation; hence there are more periods of inadequacy of the cortex, interspersed with periods of recovery. In the directing of the slow component a canceling out of the two factors producing slow movement toward center leaves the cortex better able to maintain deviation. When the frontal cortex is not adequate, the reflex retinal system producing fixation becomes accentuated. This modifies ocular movement.

(The method for recording movements of the eye was explained, and the results of some experiments were described and illustrated by lantern slides. The organization plan of the movie was described, starting with the eye in central position, free of cortical control.)

DISCUSSION

DR. SHERMAN L. SHAPIRO: There was an apparatus, used, I believe, by Benjamin, for measuring movements with glasses, occluded glasses with markings on them, something like the Bartels glasses. If the contrarolling movements take place with fixation out of the reckoning, how can Dr. Perlman say it is due to occipital reflex?

DR. HENRY PERLMAN: The first work on this problem was done by physiologists and psychologists, who demonstrated that it was possible to use such a method for recording ocular movements. It seemed an opportunity for otologists to study some of the clinical problems in this field, using the new method.

The apparatus which was used in this study was an A. C. condenser-coupled amplifier with a wide range of frequency response and a high gain; 60 microvolts produced about a quarter-inch displacement of the pen. When one records with an ink writer one is of course limited by its physical properties. But the resonance frequency of the ink writer was about 100 per second, quite adequate for recording these relatively slow movements of the eye. There is one fault with this kind of

amplifier: The displacement of the needle is dependent on the decay of the condensers. The time constant represents one-half the period for total decay and is about 0.8 second. As was noted in the records, the eye in the maintained deviated position does not keep the pen of the ink writer constantly deviated from center. The pen gradually recedes toward center. That represents the decay of the amplifier. However, practically all the phenomena one wishes to record are faster than 0.8 second.

All these records were made with the assistance of Dr. Case, who is in charge of the laboratory of electroencephalography. From our records it appears that regular countrarolling movements of the eye do not occur with the eye closed. The eyes behave differently when open, even behind strong lenses. As long as there is retinal stimulation something happens that makes the open eye behave differently from the closed eye. We feel sure that we are dealing with obliteration of the retinal system when the eye is closed.

Book Reviews

Diseases of the Nose, Throat and Ear. Edited by Chevalier Jackson, M.D., Sc.D., LL.D., and Chevalier L. Jackson, M.D. Pp. 844, with 934 illustrations on 581 figures, including 18 plates in color. Price $10. Philadelphia: W. B. Saunders Company, 1945.

The volume is made up of contributions from an impressive list of men, all authorities in their respective fields. The material is divided into the obvious divisions of otolaryngology with the addition of excellent sections on the trachea, the bronchi and the esophagus and on foreign bodies in the air and the food passages. In addition to these, there are sections on anesthesia, surgery in tuberculous patients, photography, chemotherapy, aviation otolaryngology and other pertinent subjects. The anatomy and the physiology of the various parts are well covered, and infectious, allergic, cancerous and traumatic involvements of the different areas are described and discussed. Many procedures of treatment are given in detail and with excellent illustrations, some of which are in color. Altogether, the volume is well conceived and well and readably written. It should be a valuable addition to one's library.

Directory of Otolaryngologic Societies *

INTERNATIONAL

First Pan-American Congress of Oto-Rhino-Laryngology and Bronchoesophagology

Predident: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Chevalier L. Jackson, 255 S. 17th St., Philadelphia 3.
Place: Palmer House, Chicago. Time: Oct. 17-19, 1946.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Secretary: Dr. Fletcher D. Woodward, 104 E. Market St., Charlottesville, Va.
Place: San Francisco. Time: July 1-5, 1946.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
President-Elect: Dr. Alan C. Woods, Johns Hopkins Hospital, Baltimore 5.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100–1st Ave. Bldg., Rochester, Minn.

American Broncho-Esophagological Association

President: Dr. Carlos E. Pitkin, Carnegie Medical Bldg., Cleveland.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago 11.
Place: Drake Hotel, Chicago. Time: May 31, 1946.

American Laryngological Association

President: Dr. Harold I. Lillie, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis.
Place: Drake Hotel, Chicago. Time: May 29-30, 1946.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Albert C. Furstenberg, University Hospital, Ann Arbor, Mich.
Secretary. Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Drake Hotel, Chicago. Time: May 27-28, 1946.

SECTIONS:

Eastern.—Chairman: Dr. Philip E. Meltzer, 20 Charlesgate West, Boston 15.
Southern.—Chairman: Dr. E. Trible Gatewood, Professional Bldg., Richmond 19, Va.
Middle.—Chairman: Dr. Lawrence R. Boies, Medical Arts Bldg., Minneapolis 2.
Western.—Chairman: Dr. Clarence A. Veasey Jr., Paulsen Bldg., Spokane 8, Wash.

American Otological Society

President: Dr. Gordon Berry, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Gordon D. Hoople, 713 E. Genesee St., Syracuse, N. Y.
Time: May 31-June 1, 1946.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

President: Dr. Alfred Schattner, 115 E. 61st St., New York.
Secretary: Dr. Norman N. Smith, 291 Whitney Ave., New Haven, Conn.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.